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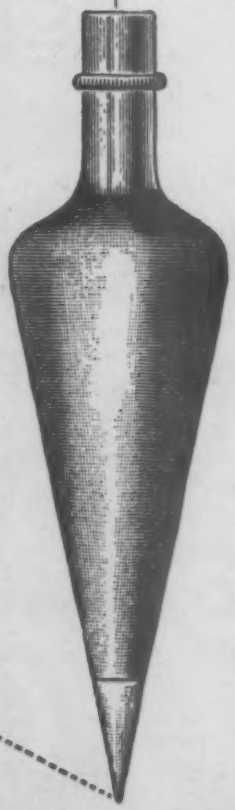

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COMPARTMENTAL DISTRIBUTION OF SODIUM CHLORIDE IN SURGICAL PATIENTS PRE- AND POSTOPERATIVELY*

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NORMAL INDIVIDUALS usually tolerate large amounts of intravenously administered isotonic sodium chloride. Those same persons, however, during the immediate postoperative period may develop rather serious complications from relatively slight excesses of saline solutions. The observation that surgical patients developed salt intolerance had been stated at the turn of the century by Evans (1911),[1] Trout (1913)[2] and later by Matas (1924).[3] In subsequent years, however, emphasis was directed to the complications resulting from salt loss, and an era of postoperative saline administration ensued.[4-10] Coller, *et al.* (1938) formulated a "clinical rule" aimed at quantitatively replacing depleted saline in surgical patients.[11]

Stimulated by the studies concerning the dangers of chloride deprivation, many surgeons administered saline solution regardless of whether or not losses had occurred, and frequently excessive quantities of sodium chloride solution were administered. It was natural, accordingly, for symptoms of salt retention to become manifest and for rather dangerous clinical sequelae to develop. Since then reports have appeared cautioning against promiscuous administration of saline solutions,[12, 13] focusing attention on the fact that the sick surgical patient does not tolerate excess sodium chloride[9, 14] and directing efforts to determine criteria for the quantity and type of fluid to administer postoperatively.[15] Wangensteen in 1942 stressed the danger of uncontrolled water and salt administration postoperatively and described a method of determining the fluid status of patients following surgery by gravimetric means.[13]

Coller, *et al.*[16] in 1944 retracted the so-called clinical rule for chloride administration and stated that no isotonic saline solution or Ringer's solution should be given during the day of operation and during the subsequent two postoperative days.

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Because controlled electrolytic and fluid administration is germaine to uncomplicated postoperative convalescence, and since either hypochloremia or excessive chloride administration contribute to the development of clinical complications, it was considered pertinent to investigate the manner by which the body handles a load of sodium chloride. An equal amount of sodium chloride given to the same patients preoperatively and shortly after they were subjected to surgical intervention would permit a comparison of the metabolism of the salt pre- and postoperatively and possibly point out the mechanics of the postoperative salt intolerance.

METHODS OF STUDY

A salt tolerance test was utilized which permitted the observance of the metabolism of a load of sodium chloride over a period of 24 hours. All tests were performed according to the following routine. The patients were permitted no food during the test period, but salt-free oral fluid was permitted ad lib. The morning of the test (usually three to five days preceding surgery) the morning voided urine was discarded. The patient was then weighed and samples of venous blood withdrawn for the following determinations: serum protein, chloride, carbon dioxide combining power, sodium and hematocrit. Blood samples were obtained also in heparinized syringes as blanks for determining plasma volume and extracellular space. Three milliliters of 0.5 per cent solution of Evans Blue Dye and 20 ml. of 5 per cent sodium thiocyanate were injected intravenously for plasma volume and thiocyanate space determination. Twenty minutes after the injection, blood samples were again taken from the opposite antecubital vein for measurement of the dye dilution.

The patient remained in bed, and an intravenous infusion of 27 Gm. of sodium chloride dissolved in three liters of 5 per cent dextrose and water was administered. An average of four hours was required for the fluid to be given.

Immediately after the completion of the infusion the patients were again weighed, blood specimens withdrawn for chemical determination, and the urine was collected during this period via an indwelling Foley catheter within the bladder, and analyzed for chloride content. The chemical analyses of the blood and plasma volume and thiocyanate (available fluid) space were performed again three hours after the completion of the infusion. Urine specimens during this three hour period were measured for total volume and chloride content. Blood and urine analyses were performed again 24 hours after the completion of the infusion (21 hours after above urine collection). It was felt that by this method a continuous record of the manner in which the organism handled the 27 Gm. of sodium chloride for the 24-hour period could be ascertained.

From the serum concentration of protein and chloride and the plasma volume and thiocyanate space one can determine the total quantity of circulating protein and the total distribution of the chloride ion within the plasma and interstitial space (the thiocyanate space theoretically measures the extra-

cellular compartment, which represents the space in which chloride is distributed). In this presentation the plasma volume is subtracted from the thiocyanate volume to permit an evaluation of the interstitial volume. A comparison of the total circulating serum chloride and the total chloride content of the interstitial spaces pre- and postoperatively is thus permitted.

The salt tolerance test was repeated again on the first postoperative day. In two patients (Table I, patients 1 and 4) the salt tolerance test was performed on the second postoperative day, since it was felt that 27 Gm. of sodium chloride might be dangerous because of the precarious clinical state of the patient.

General inhalation anesthesia was given to each patient, as well as preanesthetic opiate and barbital. Blood and fluid volume was usually maintained by administration of appropriate solutions during surgery. No saline was given postoperatively except the test dose herein described.

CHEMICAL METHODS AND CALCULATIONS

Serum chloride was performed by the technic of Schales and Schales.[17] Serum protein concentrations were determined by the method of Weichselbaum;[18] serum sodium by an internally compensated Perkin-Elmer flame photometer, and the hematocrit as described by Musser and Weintrobe.[19] The carbon dioxide combining capacity was determined by the gasometric method of Van Slyke and Cullen.[20] Urine chloride concentration was ascertained by the modified Volhard-Harvey titration method.[20] Plasma volume and thiocyanate (available fluid) space was measured by the procedure developed by Gregersen and Stewart[21] adapted for the Evelyn Colorimeter. One postinjection sample was obtained, since Noble and Gregersen have demonstrated that results so obtained vary but little from the method of obtaining several blood samples and projecting the dilution values to zero time.[22] The 20 minute postinjection period for withdrawal of the blood for measurement of dye dilution was taken as the most convenient point to withdraw samples for both T1824 and thiocyanate determination. Noble and Gregersen have shown that at 20 minutes after injection the variation from the multiple sample method is only 1 to 5 per cent.[22] The thiocyanate disappearance continues quite steadily for approximately 30 minutes.[23] At 20 minutes, therefore, a compromise between the above values is achieved which permits estimation of plasma and thiocyanate volume simultaneously. Although these total values may differ slightly from those obtained by multiple sample technics, the method was used to compare results in the same patient at different times, and therefore comparison of values in the same patient in the different states is permitted. Total circulating plasma proteins were calculated by multiplying the plasma protein concentration with the plasma volume. Total serum water chloride was calculated by determining the chloride concentration in serum water and multiplying that value by the total serum water.[24]

The interstitial chloride volume was calculated by multiplying the interstitial volume (obtained by subtracting the plasma volume from the thiocya-

TABLE I.—*Physiologic Changes Accompanying the Intravenous Administration of 27 Gm. of Sodium Chloride in 3000 Ml. of a 5 Per Cent Solution of Dextrose Given Pre- and Postoperatively.*

Case Sex Age	Diagnosis and Operation	Time (hrs.) after Infusion	Weight (Kg.)	Hematocrit Red Blood Cells (%)	Serum Protein (Gm. %)	Serum Chloride mEq. per liter	Serum Sodium mEq. per liter	Urine** Chloride (Gm./L.)	Urine Volume (ml.)	Total Urine Chloride (Gm.)	Total Fluid Intake (ml.)	Fluid Loss Drainage (ml.)
		Preoperatively										
1. P. F.* F—41	Generalized polyposis of colon	Pre	78.8	40.0	6.9	109						
		Immed.		39.0		116		11.3	325	3.7	3400	
	Total colectomy	3 hrs.	79.6	38.0	7.2	116		4.1	152	0.6		
		24 hrs.	78.8	43.0	7.0	112		8.2	1650	13.5		
		Postoperatively										
		Pre	76.2	37.0		91						
		Immed.		33.0		101		1.5	300	3.4		
		3 hrs.	77.4	35.0		109		1.6	200	0.3	4400	850
		24 hrs.	76.3	37.0	5.9	102		1.0	3400	3.2		
		Preoperatively										
2. N. B. M—35	Cardiospasm	Pre		44.2	6.9	105						
		Immed.		43.0	6.9	115		4.0	410	1.6	4000	
	Esophagogastrectomy	3 hrs.		41.2	8.0	118		8.9	550	4.9		
		24 hrs.		44.5	8.8	110		5.9	2120	12.5		
		Postoperatively										
		Pre	69.2	49.0		98						
		Immed.				107		6.7	415	2.8	3000	
		3 hrs.	69.4			107		5.5	600	3.3		
		24 hrs.		42.0		98		1.1	1040	1.2		
		Preoperatively										
3. I. R. F—61	Carcinoma of the stomach	Pre	61.8	30.3	6.2	110						
		Immed.		25.5	5.7	119		7.1	205	1.44	3500	
	Gastrojejunostomy and splenectomy	3 hrs.	64.0	25.0	5.7	121		13.5	56	0.76		
		24 hrs.	63.2	28.0	6.7	114		11.2	790	8.9		
		Postoperatively										
		Pre	62.2	56.0	6.3	101						
		Immed.		47.0	5.2	108		5.5	340	0.6	4300	1200
		3 hrs.	64.0	47.0	5.2	105		5.5	120	0.6		
		24 hrs.	62.0	50.0	5.5	106		3.5	930	2.4		
		Preoperatively										
4. H. L.* M—65	Small bowel obstruction	Pre	62.6	48.8	6.8	105						
		Immed.		44.0		119		3.8	195	0.7	4700	
	Enterolysis	3 hrs.	64.0	41.0	6.8	112		5.1	201	1.0		
		24 hrs.		41.0	7.0	100		3.7	630	2.3		
		Postoperatively										
		Pre	62.2	43.0	6.5	100						
		Immed.		41.7		109		3.1	35	0.7	4600	325
		3 hrs.	64.0	41.0	5.6	107		1.2	1195	1.4		
		24 hrs.	62.2	44.5		110		2.5	1770	4.4		

TABLE I.—*Physiologic Changes Accompanying the Intravenous Administration of 27 Gm. of Sodium Chloride in 3000 Ml. of a 5 Per Cent Solution of Dextrose Given Pre- and Postoperatively—(Continued).*

Case Sex Age	Diagnosis and Operation	Time (hrs.) after Infusion	Weight (Kg.)	Hematocrit Red Blood Cells (%)	Serum Protein (Gm. %)	Serum Chloride mEq. per liter	Serum Sodium mEq. per liter	Urine** Chloride (Gm./L.)	Urine Volume (ml.)	Total Urine Chloride (Gm.)	Total Fluid Intake (ml.)	Fluid Loss Drainage (ml.)
		Preoperatively										
5. I. W. M—73	Carcinoma of the rectum	Pre	47.0	47.5	7.0	105						
		Immed.		50.4	6.7	113		6.0	1300	7.8	3000	
	Abdominal-perineal resection	3 hrs.	48.8	43.7	7.0	108		10.1	125	1.3		
		24 hrs.		45.0		108		8.2	980	8.0		
		Postoperatively										
		Pre	48.5	53.4	6.9	98						
		Immed.			5.4	112		1.7	550	1.0	4000	500
		3 hrs.	49.4	47.0	5.6	103		2.9	930	2.7		
		24 hrs.		47.8		101		3.1	1516	4.7		
		Preoperatively										
6. J. P. M—61	Carcinoma of the stomach	Pre	62.0	42.0	6.9	104	137					
		Immed.		35.3	6.3	114	150	6.4	420	2.7	4000	
	Gastrectomy (partial)	3 hrs.	64.2	38.5	7.5	112	144	7.8	430	3.3		
		24 hrs.	61.6	31.5	6.5	103	141	5.6	2780	15.1		
		Postoperatively										
		Pre	61.0	45.0	6.1	103	132					
		Postoperatively										
		Pre	61.0	45.0	6.1	103	132					
		Immed.		43.5	6.0	104	138	6.0	645	3.9	4000	1000
		3 hrs.	62.8	42.5	6.1	103	138	9.7	200	1.9		
		24 hrs.	61.4	39.0	6.0	101	139	5.3	460	2.4		
		Preoperatively										
7. C. E. M—23	Ulcerative colitis	Pre	50.6	45.6	7.6	102						
		Immed.		40.5	6.8	111		11.5	120	1.4	3650	
	Ileostomy	3 hrs.			6.8	101		16.2	88	1.4		
		24 hrs.	53.6	39.0		100		7.1	1200	8.5		
		Postoperatively										
		Pre	52.6	47.5	7.9	93						
		Immed.		39.0	6.3	109		2.5	785	1.9	4150	1300
		3 hrs.		41.0	6.3	106						
		24 hrs.	52.1	44.5		99		11.0	500	6.0		

(Continued on Page 1014)

TABLE I.—*Physiologic Changes Accompanying the Intravenous Administration of 27 Gm. of Sodium Chloride in 3000 Ml. of a 5 Per Cent Solution of Dextrose Given Pre- and Postoperatively—(Continued).*

Case Sex Age	Diagnosis and Operation	Time (hrs.) after Infusion	Weight (Kg.)	Hematocrit Red Blood Cells (%)	Serum Protein (Gm. %)	Serum Chloride mEq. per liter	Serum Sodium mEq. per liter	Urine** Chloride (Gm./L.)	Urine Volume (ml.)	Total Urine Chloride (Gm.)	Total Fluid Intake (ml.)	Fluid Loss Drainage (ml.)
		Preoperatively										
8. O. F. M—75	Gastric ulcer Diabetes Subtotal gastrectomy	Pre	56.0	39.5	7.4	109	150					
		Immed.		36.0	6.9	131	156	8.0	240	1.9	4000	
		3 hrs.	56.0	36.0	6.7	122	159	8.9	230	2.1		
		24 hrs.	56.6	40.0		111	149	9.4	1050	9.9		
		Postoperatively										
		Pre	53.0	40.0	6.6	105	148					
		Immed.		34.7	7.0	108	149	2.5	640	0.6	3500	500
		3 hrs.	54.8	35.0	6.7	113	149	4.2	230	0.2		
		24 hrs.	53.0	32.7		107	149	4.6	1260	5.8		
		Preoperatively										
9. M. S. F—50	Cholecystitis	Pre	62.8	40.9	6.7	108	152					
		Immed.		34.0	5.4	119	160	5.8	690	4.0	3560	
	Cholecystectomy	3 hrs.	64.2	33.0	5.3	116	157	6.8	360	2.5		
		24 hrs.	61.6	40.5	7.2	109	154	6.0	2030	12.2		
		Postoperatively										
		Pre	60.6	43.9	6.7	100	146					
		Immed.		39.0	6.4	115	154	2.7	2360	6.3	3700	
		3 hrs.	62.2	40.3	6.2	106	150	5.9	500	2.9		
		24 hrs.	60.8	39.5	6.3	106	144	0.9	2480	2.2		
		Preoperatively										
10. S. D. F—27	Cholecystitis	Pre	58.2	33.9	7.6	105	144					
		Immed.		31.7	6.8	115		5.1	2070	10.5	3000	
	Cholecystectomy	3 hrs.	59.0	38.9	6.4	108	147	10.1	290	2.8		
		24 hrs.		34.0	6.1	104	154					
		Postoperatively										
		Pre	57.6	43.8	7.0	99	137					
		Immed.		37.0	6.6	110		0.6	1120	0.6	4000	380
		3 hrs.	58.0	37.8	6.8	104	138	2.8	730	2.1		
		24 hrs.	56.4	37.0	6.8	104	137	9.2	955	8.8		

* Salt tolerance test performed on the second postoperative day.
** Expressed as sodium chloride.

nate volume) by the extracellular chloride concentration[24] (serum water chloride concentration corrected for Gibbs Donnan effect).

RESULTS

Salt tolerance tests were performed on ten patients pre- and postoperatively. In seven patients plasma volume and thiocyanate space were determined permitting the calculations of total plasma and interstitial volumes and chloride content. The data are presented in Tables I and II. The results can be expressed best by describing the values obtained by each component part of this study.

TABLE II.—*Effects of Three Liters of 0.9 Per Cent Sodium Chloride Solution Administered Intravenously, Pre- and Postoperatively, Upon Plasma Volume, Interstitial Volume, Total Circulating Serum Protein, Total Serum Chloride and Total Interstitial Chloride.*

			Plasma Volume (Liters)	Interstitial Volume (Liters)	Total Circulating Serum Protein (Grams)	Total Serum Chloride (mEq.)	Total Interstitial Chloride (mEq.)
Case 3 I. R.	Preop.	Pre I. V.	2.5	11.6	155	274	1392
		Post I. V.	3.8	12.5	217	459	1391
	Postop.	Pre I. V.	2.6	12.5	164	263	1425
		Post I. V.	3.2	14.5	166	337	1696
Case 4 H. L.	Preop.	Pre I. V.	3.0	14.0	204	316	1960
		Post I. V.	4.2	14.1	286	470	2516
	Postop.	Pre I. V.	3.3	12.8	214	223	1446
		Post I. V.	4.0	14.3	224	427	1716
Case 5 I. W.	Preop.	Pre I. V.	2.2	10.6	154	231	1403
		Post I. V.	3.5	11.5	245	378	1523
	Postop.	Pre I. V.	2.5	10.0	172	244	1110
		Post I. V.	3.0	12.8	171	308	1427
Case 7 C. E.	Preop.	Pre I. V.	2.2	12.0	167	224	1212
		Post I. V.	4.0	12.2	272	403	1561
	Postop.	Pre I. V.	3.0	13.4	219	281	1420
		Post I. V.	3.6	15.0	227	381	1785
Case 8 O. F.	Preop.	Pre I. V.	3.4	15.8	252	371	1427
		Post I. V.	414	18.1	254	542	1688
	Postop.	Pre I. V.	3.5	16.5	231	366	1943
		Post I. V.	4.0	17.7	268	452	2240
Case 9 M. S.	Preop.	Pre I. V.	3.4	11.5	228	368	1666
		Post I. V.	4.8	11.9	254	555	1777
	Postop.	Pre I. V.	3.5	12.5	234	349	1412
		Post I. V.	3.9	14.5	242	413	1725
Case 10 S. D.	Preop.	Pre I. V.	2.6	10.1	198	274	1272
		Post I. V.	3.8	12.9	243	409	1403
	Postop.	Pre I. V.	3.0	10.4	210	296	1165
		Post I. V.	3.8	12.8	258	397	1510

Serum Chloride Concentration. Figure 1 presents the percentage increase of the serum chloride concentration at various times following the intravenous injection of 27 Gm. of sodium chloride. During the preoperative studies it may be noted that immediately after the completion of the infusion the average increase of the serum chloride concentration is 10.3 per cent above the preinfusion value of 106 mEq. per liter. Following this peak during the subsequent three hours there is a gradual drop to an average 4.7 per cent increase

above the pre-infusion level. During the subsequent 21 hours the descent of the serum chloride concentration continues and approaches the pre-infusion value (averaging 107 mEq. per liter).

Postoperatively a somewhat different pattern of response to the 27 Gm. of sodium chloride is reflected by the serum chloride concentration at varying time intervals subsequent to its injection. The peak of the serum chloride level is increased to 9.5 per cent above the pre-infusion level and the drop is more gradual, so that at three hours the average value is 7.8 per cent above the

TABLE III.—*Alteration of Plasma Volume and Total Serum Chloride in Seven Patients Subsequent to the Administration of 27 Gm. of Sodium Chloride (463 mEq. of Chloride) During the Pre- and Postoperative Period.*

	Preoperative	Postoperative
Average plasma volume (liters) preinfusion	2.77	3.0
Average plasma volume (liters) 3 hours postinfusion	4.07	3.64
% increase of plasma volume	46.9	21.3
Average serum chloride (milliequivalents) preinfusion	294	289
Average serum chloride (milliequivalents) 3 hours postinfusion	459	344
% increase of serum chloride	56.1	19

pre-infusion level, and it remains 4.9 per cent above the initial concentration 24 hours subsequent to the injection.[2]

A diminished hematocrit during the postinfusion periods reflected the hemodilution. A diminution of serum protein concentration occurred usually, but no characteristic pattern of response was observed.

The serum concentration of sodium at the different periods subsequent to the saline injection was determined in four patients, and the distribution simulates that exhibited by the chloride ion.

TABLE IV.—*Alteration of Interstitial Volume and Total Interstitial Chloride in Seven Patients Subsequent to the Administration of 27 Gm. of Sodium Chloride (463 mEq. of Chloride) During the Pre- and Postoperative Period.*

	Preoperative	Postoperative
Average interstitial volume (liters) preinfusion	12.2	12.4
Average interstitial volume (liters) 3 hours postinfusion	13.3	14.5
% increase of interstitial volume	9	16.1
Average interstitial chloride (milliequivalents) preinfusion	1476	1417
Average interstitial chloride (milliequivalents) 3 hours post-infusion	1694	1728
% increase of interstitial chloride	14.7	21.9

Alterations in Plasma Volume and Total Serum Chloride. In order to determine whether any difference exists in the total serum chloride preoperatively from that during the postoperative period analyses of total volume in both periods were compared. Table III reveals that three hours after the infusion, before the patient had been subjected to surgery, the plasma volume was increased an average of 46.9 per cent above the preinfusion level of 2.77 liters. Associated with the increased plasma volume was an increase of the

total serum water chloride of 56.1 per cent above the preinfusion total serum water chloride value of 294 mEq. Subsequent to surgical intervention, the plasma volume, which was approximately the same as that observed preoperatively (averaged 3.0 liters), increased but 21.3 per cent following the administration of 3 liters of fluid containing 27 Gm. of sodium chloride. Associated

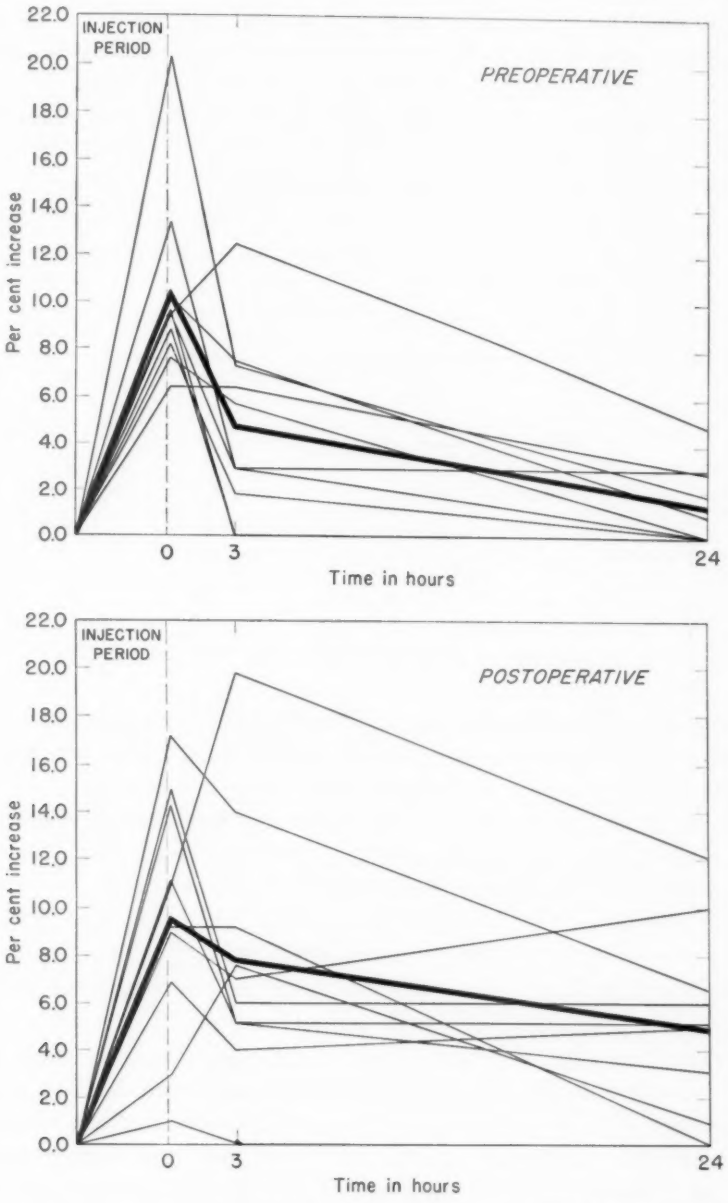

FIG. 1.—The percentage increase of the serum chloride at varying time intervals following the intravenous administration of 27 Gm. of sodium chloride in 3 liters of 5 per cent dextrose in distilled water

with the lesser expansion of the plasma volume was noted a lesser increase of total serum water chloride postoperatively (19 per cent).

These results imply that postoperatively less saline is retained in the circulatory compartment following the administration of 3 liters of normal saline solution and suggest that the compartmental distribution of electrolyte might be altered. This premise has been substantiated by the following data.

Alterations in Interstitial Volume and Total Interstitial Chloride. Table IV summarizes the average changes which occurred in the interstitial volume and the total interstitial chloride subsequent to the administration of 27 Gm. of sodium chloride as normal saline pre- and postoperatively. The average interstitial volumes were similar during the pre- and postoperative periods before the intravenous salt solution was administered. Three hours after the completion of the infusion the average interstitial volume had expanded 9 per cent during the preoperative study, while the expansion was greater postoperatively, averaging 16.1 per cent. Similarly, an increased amount of chloride was present in the interstitial space postoperatively. The average postop-

TABLE V.—*Alteration of Total Circulating Serum Protein in Seven Patients Subsequent to the Administration of 27 Gm. of Sodium Chloride in 3 Liters of Fluid During the Pre- and Postoperative Period.*

	Preoperative	Postoperative
Average total serum protein (Grams) preinfusion	194.0	206.0
Average total serum protein (Grams) postinfusion	253.0	221.0
Average % increase	30.4	7.3

erative interstitial chloride volume increased 21.9 per cent, whereas the average preoperative expansion of interstitial chloride mass was 14.7 per cent above the preinfusion levels of 1417 and 1476 mEq. respectively.

Effects of Saline Administration upon Total Serum Proteins. The retention of fluid and electrolytes within the circulation is linked intimately with the concentration of proteins in the serum,[26] and Stewart and Rourke have demonstrated an increased total serum protein content subsequent to saline infusions, which they feel permits circulation of the extracellular fluid between the vascular and interstitial compartments.[27] For this reason the total serum proteins of the patients in this study were determined pre- and postoperatively preceding and three hours after the completion of the saline infusion. The data are summarized in Table V and reveal an average increase in the total serum protein preoperatively of 30.4 per cent above the preinfusion value of 194 Gm. Postoperatively a slight increase (7.3 per cent) occurred subsequent to the infusion. The inability of the administered saline solution to elicit a mobilization of protein into the circulation to the same degree that occurred preoperatively is possibly related to surgical trauma[28] and protein depletion, and might be a causative factor permitting the greater increase in interstitial volume and chloride mass postoperatively.

This increased deposition of chloride in the interstitial space postoperatively could enhance the retention of that ion and prevent its deliverance to

the kidneys for excretion. To investigate this hypothesis, analyses of the total urine output and its chloride content were made pre- and postoperatively.

Urinary Excretion of Chloride Following the Salt Tolerance Test. Specimens were collected during the interval of infusion, the three-hour period immediately subsequent to the termination of the infusion, and the next 21-hour period. These specimens were analyzed separately to note whether any differences in the chloride excretion occurred during the three time intervals. Table VI summarizes the urinary volume, urinary concentration of chloride and the total amount of chloride excreted subsequent to the injection of 27 Gm. of sodium chloride during the preoperative and postoperative periods.

TABLE VI.—*The Average Urinary Excretion of Chloride in Ten Patients Subsequent to the Intravenous Administration of 27 Gm. Sodium Chloride (463 mEq. of Chloride) as an Isotonic Solution Pre- and Postoperatively.*

Hours after Infusion	Preoperative	Postoperative
Average Urine Volume (Milliliters)		
Immediately	597	719
3 hours	266	522
24 hours	1470	1406
Total	2323	2647
Concentration of Chloride in Urine (Milliequivalents per liter)		
Immediately	118	56
3 hours	143	74
24 hours	114	62
Total Urinary Chloride (Milliequivalents)		
Immediately	70	40
3 hours	38	39
24 hours	167	87
Total	275	166

It may be noted that a somewhat greater total volume of urine was excreted postoperatively, as well as during each collecting period. This was somewhat unexpected because usually variable degrees of oliguria occur postoperatively.[29] However, since these tests were performed on the first postoperative day (the second postoperative day in two patients), it is probable that these particular patients had recovered from the immediate postoperative oliguric state. The total fluid intake varied but slightly, averaging 3.68 liters preoperatively and 3.97 liters postoperatively (Table I).

It is of particular interest that in the face of a rather parallel urine volume, the concentration of chloride preoperatively exceeded the concentration of urinary chloride postoperatively in each period studied. Preoperatively the urinary concentration of chloride immediately after the infusion averaged 118 mEq. per liter, while postoperatively the average was 56 mEq. per liter. During the three-hour period subsequent to the infusion, the preoperative urine chloride concentration was 143 mEq. per liter in contrast to the postoperative concentration of 74 mEq. per liter. During the subsequent 21-hour period the preoperative concentration of chloride in the urine averaged 114 mEq. per liter in contrast to 62 mEq. per liter postoperatively.

The increased urinary concentration of chloride preoperatively resulted in an increased preoperative excretion of chloride. The total amount excreted preoperatively averaged 275 mEq. (16.1 Gm. as sodium chloride) in the 24-hour period after the infusion, while postoperatively the total amount excreted in the 24-hour period averaged 166 mEq. (9.7 Gm. as sodium chloride—Table VI). A partial explanation for the diminished chloride excretion postoperatively could be the observation that eight patients lost an average of 756 ml. of fluid by means of abnormal drainage, as gastric aspiration, etc. Computing chloride loss on the basis that this fluid contains 5 Gm. of sodium chloride per liter, the postoperative patients lost an additional 65 mEq. of chloride during the entire period of study. Nevertheless, a significant difference between the two periods exists, and furthermore, during the averaged four hours necessary for the infusion to be delivered and the subsequent three hours (before a significant quantity of abnormal drainage could have exerted

TABLE *VII.—Distribution of Chloride Three Hours After the Intravenous Administration of 27 Gm. of Sodium Chloride (463 mEq. of Chloride) Pre- and Postoperatively.*

	Preoperative	Postoperative
Total increment of serum chloride (milliequivalents)	165	55
% of administered dose	35.6	11.9
Total increment of interstitial chloride (milliequivalents)	217	311
% of administered dose	46.8	67.1
Excreted chloride in urine (milliequivalents)	108	79
% of administered dose	23.3	17

an influence on the chloride metabolism), the concentration of urinary chloride postoperatively was significantly lower than that observed preoperatively, indicating some derangement of urinary chloride excretion.

This series is too small to permit a significant evaluation of the influence of the extent of the surgery, age of the patient or other factors, upon the observed phenomena. The data do suggest, however, that the extent of surgery is not a significant factor; for patient 1 (Table I), on whom a total colectomy had been performed, behaved similarly to patients 9 and 10, on whom cholecystectomies had been effected. These data suggest a somewhat greater salt retention in the higher age brackets.

DISCUSSION

The observation that during the immediate postoperative period patients do not tolerate large amounts of sodium chloride has been well documented.[9, 13-15] The sequelae which result from excess saline administration are severe[13, 30] and may contribute to the demise of the patient. Excessive salt has been administered not uncommonly, because many surgeons adopted the now obsolete clinical rule for chloride replacement of Coller[11] or attempted to correct a hypochloremia which was not the result exclusively of chloride deprivation. Other metabolic derangements, such as hemodilution,[31] hypoproteinemia,[32] or hypokalemia,[33] may result in a diminished serum chloride concentration.

In an effort to understand better the mechanism whereby an individual can normally tolerate relatively large quantities of salt solutions, but after receiving a general anesthetic and surgical intervention will retain administered saline in an abnormal manner, this series of experiments was undertaken. Attention was first directed to note the effects of anesthesia and surgery upon kidney function. Renal clearances studies were performed preoperatively and immediately after operation to study the effects of operation, including anesthesia upon renal plasma flow, glomerular filtration rate, filtration fraction and tubular excretion. Various types of alterations in the above mechanisms were observed, but in no case, except where shock had developed, could intrinsic renal dysfunction be held responsible for producing salt retention postoperatively.[34] Contrariwise, hypochloremia was observed to depress renal function.[35]

This present study was accordingly performed, utilizing a modified salt tolerance test to observe whether a prerenal chloride derangement contributed to the salt retention following surgical intervention. Soffer and his associates have utilized a three-hour salt tolerance test to study salt metabolism in Cushing's disease and the response to desoxycorticosterone acetate in normal individuals and patients with Cushing's syndrome.[36] The salt tolerance test utilized in this study was devised to study saline distribution over a longer period of time (24 hours) and in greater detail. Furthermore, it was desired to give as large a load of sodium chloride as could be tolerated safely to patients shortly after radical abdominal operations in an attempt to tax the mechanisms concerned so that derangements might be better observed.

In the preoperative studies the manner in which the salt load is distributed can be summarized as follows. The administered salt remains in the plasma for a relatively short period of time. The peak of the serum concentration of chloride, noted immediately after the completion of the infusion, decreased rapidly in the subsequent hours, and almost reached the preinfusion level 24 hours later. The test dose of water and salt three hours after its administration increased the plasma volume 46.9 per cent and the interstitial volume 9 per cent, with approximately 35.6 per cent of the administered chloride present in the serum and 46.8 per cent noted in the interstitial space.

Postoperatively the serum chloride concentration does not quite reach the same high peak as observed preoperatively, and the drop is more gradual. Twenty-four hours after the infusion it remained 4.9 per cent above the preinfusion level. In contrast to the preoperative changes a much smaller increment in volume and salt content was observed in the plasma, and a far greater retention of chloride occurred in the interstitial space, which retained 67.1 per cent of the administered chloride and expanded 2.1 liters. Expanded available fluid volumes have been reported following surgery.[37]

The somewhat increased salt mass in the interstitial space might induce hypertonicity in this compartment. If this be so, an influx of water must occur into that compartment in answer to osmotic demands, the fluid coming from the intracellular compartment.[38] To investigate the possible translocation of

fluids between the different compartments, in this experiment studies of total body water were performed utilizing the dilution technic with deuterium oxide as the tracer isotope.

Twenty-five Gm. of deuterium oxide were injected and 30 minutes later a blood sample was withdrawn. (The values may be somewhat high because it has been demonstrated that equilibrium does not occur until one hour after injection.) The serum sample was permitted to come to equilibrium with hydrogen, and the supernatant hydrogen gas was analyzed for deuterium content with the mass spectrometer. The authors are indebted to Mr. Jack Johnson

TABLE VIII.—*Distribution of Body Water Four Hours After Administration of 3 Liters 0.9 Per Cent Sodium Chloride.*

Case No. Table I	Total Body Water (Liters)	% of Body Wt.	Extra-Cellular Water* (Liters)	% of Body Wt.	Intra-Cellular Water† (Liters)	% of Body Wt.
9	32.1	50	16.7	26.0	15.4	23.0
10	33.3	57	16.7	28.3	16.6	28.1

* Thiocyanate space.

† Extracellular water subtracted from total body water.

of the Department of Physiology and Dr. Alfred Nier of the Department of Physics for these determinations. Analyses were performed upon two patients (Table I, patients 9 and 10) four hours after their infusion during the preoperative state. Table VIII presents the distribution of the body water. Total body water equals 50 per cent and 57 per cent body weight respectively. This fits in well with the findings of Dr. Francis Moore, who observed that water constituted an average of 51.9 per cent of the body weight of women (presented before Halsted Club, Minneapolis, Minnesota, December 3, 1949). It may be noted, however (Table VIII), that the water under these circumstances was more or less evenly distributed between the intra- and extracellular compartment—an abnormal situation revealing an intracellular dehydration and an interstitial edema. Since it has been demonstrated that under certain conditions the thiocyanate space is larger than the extracellular space as measured by the inulin dilution,[39] the possibility exists that a certain quantity of chloride considered interstitial may be within the intracellular compartment.[38]

Urinary studies of chloride excretion revealed an increased concentration of chloride throughout the preoperative period, with a resultant total excretion of 275 mEq. (59.4 per cent of the administered dose) 24 hours after the completion of the saline injection (including the salt excreted during the infusion period). Postoperatively 166 mEq. of chloride were excreted (35.8 per cent of the administered chloride). Since man, unlike the dog, normally lags in the excretion of administered sodium chloride,[25, 29] this data denotes that the lag is prolonged subsequent to surgical intervention.

It is interesting in this respect that Coller and his associates observed a 46 per cent retention postoperatively following the administration of a salt load similar to the one of this report given at five intervals during the 30-hour postoperative period.[16] Elman, *et al.* have noted that when postoperative patients

are given 9 Gm. of sodium chloride per day the average retention was 40 per cent.[29]

The possibility of losses occurring during operation without replacement on the surgical day could be a factor for the diminished chloride excretion; but no great disproportion of measured body chloride (plasma and interstitial chloride) between the pre- and postoperative periods was noted.

The diminution of chloride excretion subsequent to operative intervention, accordingly, might be due to the retention of the anion within the interstitial compartment. Normally saline given intravenously will be distributed between the vascular and interstitial compartment in answer to osmotic demands and the Gibbs Donnan relationship.[38] The unequal distribution observed postoperatively suggests some derangement in the normal distribution ratio. Dissociation between changes in volume of the extracellular fluid and changes in plasma volume occur following dehydration with and without salt loss.[40] Freis and Kenny have demonstrated recently that in an edematous patient with a normal pregnancy, the relationship of plasma to "available fluid" volume remains the same as normal individuals, in contrast to the eclamptic pregnant woman where a deviation from the normal relationship between the compartments occurs. There is a resultant marked disproportion between the "available fluid" which increased without a corresponding increase and not infrequently a decrease in the plasma volume.[41]

The observation that a significant quantity of protein is mobilized into the plasma (30.4 per cent increment) at a three-hour period subsequent to the infusion of 3 liters of isotonic saline solution preoperatively while subsequent to surgery a much less pronounced response (7.3 per cent increment) occurred, could be an accountable feature producing the observed alteration of fluid distribution. Stewart and Rourke have observed that an influx of serum protein following large saline infusions occurs which maintains a serum protein concentration of nearly 6 Gm. per 100 ml. They interpret this mobilization of plasma protein as a means to increase the oncotic pressure of the circulation in response to the added load of fluid and electrolyte, and to thereby maintain a physiologic relationship between the vascular and interstitial compartment.[27]

Abnormalities of fluid and electrolyte distribution are known to occur when plasma proteins are subnormal,[26, 30] and certain abnormal states, occurring postoperatively, frequently cannot be corrected until the serum protein concentration approaches normal.[32] Freis and Kenny believe the serious translocations of fluid in eclamptics to be a failure of increased plasma protein to cope with the added fluid load.[41]

The dissimilarity of protein response observed in this study to the same stimulus pre- and postoperatively might be due to a body protein loss, which is a result of surgery. It has been observed that from 10 to 20 Gm. of nitrogen are excreted per day following surgery,[28, 42, 43] which apparently represent catabolyzed tissue protein.[43-45] The possibility exists also that as a result of the anesthesia and operation, the body had been metabolically traumatized

and the mechanism which elicits the mobilization of plasma protein had been blocked.[46]

The adrenal cortex elaborates hormones capable of decreasing renal electrolyte excretion and enhancing the catabolism of protein. Salt withdrawal normally has been shown to stimulate the adrenal gland with a resultant salt retention and protein catabolism.[47] Salt administration counteracts these mechanisms mediated via the adrenal gland[2] with a resultant salt excretion and protein anabolism.[47] It is possible that the adrenal stimulation as a result of surgical trauma[46] vitiates the action normally induced by a salt load, with a resultant salt retention and blocking the body's ability to mobilize plasma protein.

In summary, this data demonstrates a different pattern of response in the distribution of chloride administered to the same individual pre- and postoperatively. In the postoperative state a larger portion is delivered to the interstitial space, and a disproportion between plasma and interstitial volume develops. Therefore, the interstitial electrolyte is apparently not as readily delivered to the kidney for excretion. Failure of plasma protein mobilization and probably the action of the adrenal gland may contribute to this mechanism.

CONCLUSIONS

1. A salt tolerance test is described which was utilized in ten patients to study the metabolism of 27 Gm. of sodium chloride administered before and after a major surgical procedure as a means of identifying the nature of postoperative salt intolerance.

2. Preoperatively the high serum chloride noted immediately after the chloride infusion decreased to the preinfusion level in 24 hours. Three hours after the infusion the plasma retained 35.6 per cent of the administered chloride, and the interstitial space retained a moderate quantity of the electrolyte (46.8 per cent). Postoperatively an increased serum chloride concentration persisted for 24 hours, a smaller amount of chloride was identified in the plasma (11.9 per cent), but a much greater quantity shifted into the interstitial space (67.1 per cent) where it was apparently retained, not being delivered readily to the kidneys for excretion.

3. The mobilization of proteins into the serum in response to the salt load preoperatively may have been a factor in retaining the material in the serum. Failure of such a response postoperatively may be an accountable feature, permitting the diffusion of chloride into the interstitial space.

4. A diminished concentration and quantity of urinary chloride was observed postoperatively compared to that noted preoperatively in each period studied.

5. Some of the mechanisms for the different distribution of chloride pre- and postoperatively and their clinical implications are discussed.

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THE SIGNIFICANCE OF PULMONARY VASCULAR LESIONS IN THE SELECTION OF PATIENTS FOR MITRAL VALVE SURGERY*

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AMONG THE PATIENTS with disabling rheumatic heart disease, a considerable number have difficulties arising almost entirely from circulatory obstruction at the mitral valve. In such patients with pure non-regurgitant mitral stenosis the most promising therapeutic approach would seem to be a direct surgical relief of the stenosis. The problem was first thoroughly investigated by Cutler, Levine, and Beck,[1] but their efforts were terminated because of the death of nine out of 11 patients operated upon. With the subsequent advances in surgical technic, control of infection, anesthesia, and pre- and postoperative care, it has seemed reasonable to reopen this problem for investigation. A much lower mortality in selected patients has already been demonstrated by Bailey and his co-workers.[2] Eighteen of their last 21 patients have survived "mitral commissurotomy." Whether their method can be applied to the severely diseased valve and whether the commissural incision will subsequently heal with recurrence of stenosis are matters that have not yet been established. At the present time technical factors such as these are overshadowed by the problem of selecting patients for operation. It is the purpose of this paper to consider the significance of coexisting pulmonary vascular lesions in candidates for mitral valve surgery.

Patients selected for mitral commissurotomy should have significant disability, reduced life expectancy, and the primarily mechanical problem of mitral obstruction. We must exclude from operation those individuals with little disability as well as those desperately ill patients with severe cardiac and pulmonary damage who have little or no chance of surviving the operation. The futility of removing the circulatory obstruction at the mitral valve in patients with severe myocardial damage, associated defects of other valves, or a large element of mitral insufficiency has been emphasized.[3] We believe it is equally futile to operate upon patients with mitral stenosis who have secondary pulmonary changes sufficiently severe to constitute a coexistent circulatory obstruction of greater degree in the pulmonary vascular bed. The possibility that certain occlusive pulmonary vascular lesions might adversely affect operative results was suggested from pathologic evidence.[7] However, the significance of this contraindication has not been given sufficient attention in the

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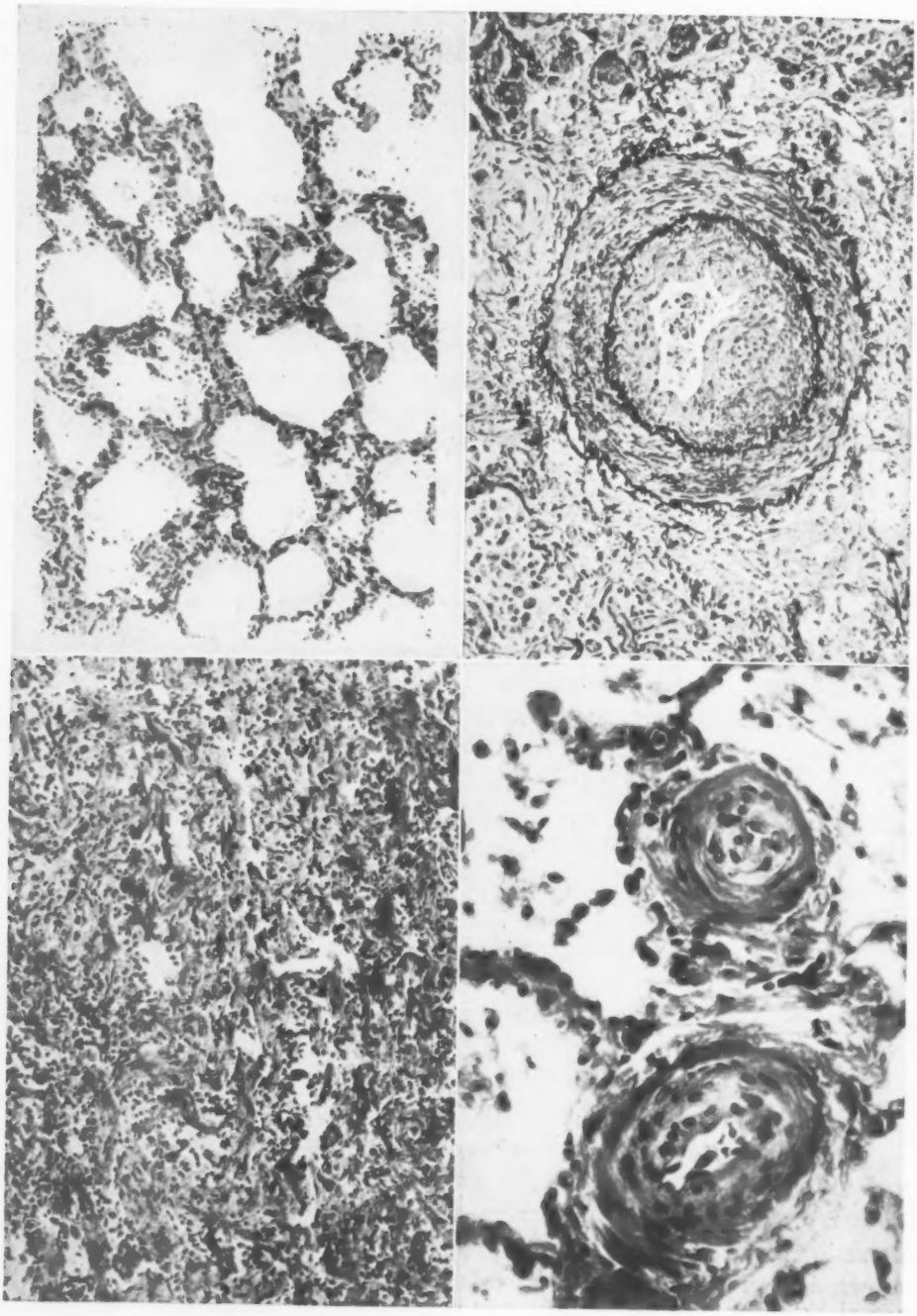

FIG. 1

FIG. 2

FIG. 3

FIG. 4

See legends on opposite page.

surgical literature to date, nor has the clinical problem of recognizing such patients been discussed.

Parker and Weiss[4] in 1936 pointed out the existence and significance of vascular lesions in the lungs which they believed to be proportional to the severity and duration of mitral obstruction. Three other series of patients have been reported calling attention to these changes.[5-7] These authors describe a lesion of the capillary basement membrane as one of the changes present in the lungs of patients with mitral stenosis. This lesion was not found in the lungs of a group of patients with congenital heart disease studied by one of us.[8] Of particular interest is the fact that this group included nine patients with combined mitral stenosis and atrioseptal defect (Lutembacher's Syndrome). These results suggest that the capillary basement membrane lesion is specific for pure mitral stenosis. It does not occur in other types of congestive failure, acyanotic congenital heart disease, pulmonary embolism, or infection. While most patients with mitral stenosis have pulmonary vascular changes,[4-7] advanced lesions are found at autopsy in only about one third. From these various observations made on pathologic study of the lungs of patients with mitral stenosis, the following sequence of events might be postulated to explain the pathogenesis of the vascular lesions: The pulmonary capillary bed lacks significant pericapillary tissue support. Hence, the increased pressure referred back from the left auricle in mitral obstruction results in a pericapillary transudate. Failure to absorb this high protein fluid from the pericapillary space results in organization and fibrosis, with thickening of the capillary basement membrane which increases at the expense of the capillary channel. Extension of the process into the septae results in diminished expansibility and loss of pulmonary reserve. This combination of events results in an increased resistance to the flow of blood through the lungs. The resulting pulmonary hypertension is associated with obliterating pathologic changes in the pulmonary arterial tree that add to this resistance. This can lead to pulmonary artery dilatation and pulmonic valve incompetence, throwing even greater burden on the right side of the heart.

It is thought that in the advanced stage these pulmonary vascular lesions result in a second circulatory obstruction in the pulmonic circuit which is in

See legends on opposite page.

FIG. 1.—Non-functioning area from the right lower lobe showing end results of chronic passive congestion. Masson's trichrome light green 80x.

FIG. 2.—Showing thickening of alveolar septal walls with pericapillary fibrosis and nearly complete obliteration of vascular channels in an area from the right middle lobe. Micrometer measurements of the thickness of septal walls in this field averaged 42μ or twice normal. Hematoxylin and Eosin 50x.

FIG. 3.—Obliterating endarteritis in a 230μ vessel and hyperplastic arteriosclerosis in a 108μ vessel. The adjacent parenchyma appears normal. This field was selected from the apical portion of the right upper lobe. Hematoxylin and Eosin 250x.

FIG. 4.—Intimal atherosclerosis in a 1 mm. vessel in the apical portion of the right lower lobe. The elastic membranes are preserved and the subendothelial connective tissue proliferation results in marked reduction of the lumen. Verhoeff-van Gieson elastic tissue 100x.

large part irreversible. The importance of this was demonstrated recently in a patient with long standing mitral obstruction who died following a technically successful commissurotomy. This case is reported as an example of a patient with mitral stenosis whose far advanced pulmonary vascular lesions caused her death despite the surgical relief of her mitral obstruction.

Case Report.—E. W., a 36-year-old white female with rheumatic heart disease was admitted to the University of Pennsylvania Hospital for mitral valve surgery. At age 11 she had her first attack of rheumatic fever. At age 22 she was found to have auricular fibrillation, and the murmur typical of mitral stenosis. At age 23 she became dyspneic and had her first bout of hemoptysis. Nine years later she had developed persistent hepatomegaly and re-accumulating ascites with dyspnea, but without orthopnea or basal râles. At the time of her present admission, she was a bedridden, cardiac invalid.

Physical examination revealed a pale, dyspneic woman, with marked venous distention. She was not orthopneic and the lungs were clear of basal râles. The cardiac rhythm was totally irregular, with a rate of 80 per minute and no pulse deficit. At the apex, the first sound was accentuated, and a grade two systolic murmur was present. In diastole there was a "mitral opening snap" followed by a grade three rumbling diastolic murmur. At the base, the second sound was accentuated and a soft diastolic murmur was present in the pulmonic area. The liver edge was firm, non-pulsating and extended 5 cm. below the costal margin. After the removal of 1300 cc. of ascitic fluid, a moderately enlarged spleen was palpable. There was no edema of the sacral or pretibial areas. Clubbing and cyanosis were not present.

Special Studies. Roentgen ray examination revealed marked cardiac enlargement, particularly of the right ventricular and left auricular chambers. The pulmonary artery was prominent and showed increased pulsations. There was no evidence of pulmonary congestion.

An electrocardiogram showed auricular fibrillation, with a ventricular pattern suggesting right ventricular hypertrophy and digitalis effects.

On cardiac catheterization, the mean pulmonary arterial pressure was found to be greater than 100 mm. of mercury. It was possible by oxygen determination and pressure readings to rule out significant complicating tricuspid stenosis, septal defect, or pericardial constriction.

Pulmonary function studies showed that the arterial oxygen saturation was 93 per cent, that the residual capacity was increased in the face of normal gas distribution, and that the vital capacity was 60 per cent of normal. These are the results usually obtained with pulmonary congestion and edema and yet basal râles were not evident clinically. Spirographic tracings suggested the existence of relatively rigid and inelastic lungs.

A bromsulphalein test showed 16 per cent retention. All other liver function studies were negative, including serum albumin and globulin, prothrombin time, cephalin flocculation, and colloidal gold test.

Hospital Course and Operative Findings. Preoperatively she was maintained on digitalis, mercurial diuretics, low salt diet and bedrest in an unsuccessful attempt to relieve the ascites, hepatomegaly, and elevated venous pressure. An antero-lateral mitral commissurotomy[2] was performed, with the feeling that this represented her only chance of relief. A striking finding at operation was that of an enormous pulmonary artery with a pressure that, by palpation, seemed almost equal to that of the much smaller aorta. After commissurotomy the pressure in the pulmonary artery did not change appreciably to palpation.

Postoperatively, absence of the "mitral opening snap" and of the mitral diastolic murmur suggested satisfactory relief of the valvular obstruction. However, systemic hypotension developed and the venous pressure rose in the absence of significant postoperative pulmonary complications or physical signs of pulmonary congestion (*i.e.*, basal

râles). With this clinical picture it seemed likely that she had an obstruction in her pulmonary vascular bed accounting for the absence of râles, failure of filling of the left side of the heart, and diminished cardiac output. The systemic hypotension and elevated venous pressure continued until the time of her death 3 days later.

AUTOPSY FINDINGS

Except for ascites, and chronic passive congestion of the liver and spleen, the principal findings were limited to the heart and lungs.

Heart—Gross. The heart was twice normal in size and weighed 640 Gm. This enlargement was predominantly right-sided. On opening the surgical incision in the left auricular appendage, the interauricular septum was found to be intact and there was a typical "fishmouth" deformity of the mitral valve. The original opening measured only 8 mm. in diameter. The commissurotomy incision appeared satisfactory, being anterolateral, 1.3 cm. in length, and showing no evidence of healing. The valve was sufficiently pliable so that the incision apparently had relieved the stenosis. There was minimal rheumatic involvement of the aortic valve. The cut surface of the myocardium appeared normal and the coronary vessels were patent. The right ventricular wall measured 1.0 cm. in thickness, and the left 1.5 cm. The main pulmonary artery showed atherosclerotic change and measured 9.5 cm. in circumference 2.0 cm. distal to the valve. This was nearly twice the circumference of the aorta at the same level.

Heart—Microscopic. There were numerous foci of scarring in the myocardium. However, there was no evidence of active rheumatic myocarditis.

Lungs—Gross. On cut surface, the sclerotic smaller branches of the pulmonary artery stood out sharply and there was evidence of "brown induration" with superimposed terminal congestion.

Lungs—Microscopic. (1) Method: Several blocks of tissue were taken from each lobe, and were fixed in 10 per cent Formalin. Sections from these blocks were stained with hematoxylin and eosin, a combination of Van Gieson's and Verhoeff's elastic tissue method on the same section, and Masson's trichrome light green. The vascular changes encountered were so striking that it seemed superfluous to make the calculation of wall to lumen ratios recommended by Kernohan, *et al.*[9] An ocular screw micrometer was used to measure the external diameter and medial coats of vessels and to determine to what degree the capillary basement membrane lesion of Parker and Weiss was present.

(2) Findings: The alveolar septal lesion described by Parker and Weiss[4] varied in severity but was uniformly present in the lower two thirds of each lung. In these areas fibrosis was marked and the normal architectural pattern was destroyed (see Fig. 1). Above this level there was a gradual change to a coarse latticed structure with marked thickening of the alveolar septae due to collagen deposition. This amorphous material compressed or obliterated alveolar capillary channels and separated them from air filled spaces. Such an area is included in Figure 2.

In addition to the pericapillary lesions, severe and constant obliterating vascular lesions were encountered throughout all lobes of the lungs. The arterioles showed a laminated sclerosis of the type commonly seen in the systemic arteries in hypertension. The lumina of these vessels were often reduced to capillary size. Obliterating endarteritis was consistently found in the small arteries. Figure 3 shows both of these lesions in a section taken from the apical portion of the right upper lobe. Grossly this part of the lung appeared normal. Marked reduction in lumen is seen in a 1 mm. vessel in Figure 4. In all of the arteries greater than 1 mm. in external diameter which were examined microscopically, severe atherosclerotic changes were found. Changes in the medial coat of vessels were not impressive in the Verhoeff/van Geison preparations. The type of arteritis described by Von Glahn and Pappenheimer in cases of active rheumatic infection was not found.[10] There was no evidence of extensive thrombosis with reorganization. The pulmonary veins appeared normal but were of large size.

DISCUSSION

Despite a technically successful commissurotomy this patient died with severe hypotension and remarkable engorgement of the peripheral veins and liver. We believe that she died in spite of relieving the mitral stenosis, because there was a coexistent obstruction of a greater degree in the pulmonary circulation. The vascular changes in the lungs found on pathologic examination seemed to explain her inability to fill adequately the left side of the heart.

During the present developmental phase of mitral valve surgery, it is probably inevitable that most of the patients with mitral stenosis subjected to operation will be those whose prognosis is otherwise poor. It is important, therefore, to attempt to recognize and to exclude individuals who will not be helped by operation. The patient reported above represents one type of contraindication; namely, the individual with pulmonary vascular obstruction of a degree sufficiently severe to make it relatively useless to relieve the mitral stenosis. The following characteristics shown by this patient deserve emphasis and might allow preoperative recognition of this contraindication in the future:

1. A history of disability of long duration was obtained in a patient with known mitral stenosis.

2. Long-standing signs of venous distention, hepatomegaly, and ascites were present and were not cleared up by medical treatment.

3. Severe dyspnea occurred on effort, but significant orthopnea and basal râles were not present.

4. An unusually large and hyperpulsatile pulmonary artery was observed fluoroscopically and was considered suggestive of hypertension within the pulmonary circulation.

5. A markedly elevated pulmonary artery pressure was found on cardiac catheterization. The mean pressure was over 100 mm. Hg., even though the reading was made after a prolonged period of treatment directed towards relief of cardiac failure. Studies of a series of patients will be necessary before a range of pressure can be determined above which operation is likely to fail. Serial cardiac catheterization has been shown to offer information about changes in pulmonary artery flow as well as the degree and lability of pulmonary hypertension.[11, 12]

6. Pulmonary function studies revealed a diminished arterial oxygen saturation and delay in the oxygen diffusion rate.*

* Pulmonary function studies have been somewhat disappointing thus far in helping to assess these patients preoperatively. It should be possible theoretically to estimate the severity of the alveolar septal lesion by determining the functional state of the alveolocapillary membrane. Unfortunately the alteration in oxygen diffusion rate expected with pericapillary fibrosis occurs in a number of other conditions. Such changes must therefore be interpreted in the light of other findings, such as the presence or absence of râles. Spirographic tracings probably give more information as to the existence of parenchymal and vascular damage.

The above points may help to characterize the individual with severe pulmonary vascular changes. This type of patient must be distinguished from individuals with mitral stenosis complicated by other lesions of congenital or rheumatic origin, such as tricuspid disease, septal defects, or constrictive pericarditis. Although ability to recognize such severely handicapped patients is helpful, we are still faced with the problem of how severe pulmonary changes may be and yet permit the patient to be improved following surgical release of the mitral obstruction. Our tentative feeling is that a patient does not have pulmonary vascular lesions of sufficient severity to contraindicate surgery if (1) he can be freed of failure on medical treatment, (2) the venous pressure and arm to lung circulation time become normal, and (3) the pulmonary artery pressure is not fixed at a markedly elevated level.

At present, no combination of laboratory tests infallibly assesses the degree and extent of obstructing pulmonary vascular changes. It has been demonstrated that quantitative evaluation of vascular change is possible by establishing the resistance to flow and pressure gradient across the pulmonary capillary bed.[13] Direct pressure measurements of this general type could be obtained at the time of operation in patients with mitral stenosis. In the event of a markedly increased pressure gradient between the pulmonary artery and vein, one would anticipate little improvement after operation upon the mitral valve. However, reversibility of vascular and parenchymal damage may be possible to some degree.

CONCLUSION

1. Pulmonary vascular lesions may produce secondary obstruction within the pulmonic circuit in patients with long standing mitral stenosis.

2. The significance of these pulmonary vascular lesions in the selection of patients for mitral valve surgery has been given only scant attention in the surgical literature.

3. A patient with mitral stenosis is described in whom pulmonary vascular lesions were present to a marked degree. These seemed to account for failure of the mechanically adequate mitral commissurotomy to relieve the existing circulatory obstruction.

4. Previous observations and a review of the literature suggest that such patients will not derive benefit from surgical measures aimed at the relief of mitral stenosis.

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AORTIC RESECTION AND ANASTOMOSIS IN PUPS STUDIED AFTER REACHING ADULTHOOD*

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ON OCTOBER 19, 1944, Crafoord of Stockholm, Sweden,[1] performed an exploratory thoracotomy on a patient with coarctation of the aorta to see if surgical correction of this congenital anomaly was feasible. He found he was able to resect the stenosed portion of the thoracic aorta, and re-establish the continuity of the vessel by direct end-to-end anastomosis of the proximal and distal segments. This was accomplished by using Carrel's method of arterial suture, thus approximating the various layers by sutures which penetrated the adventitia and media, but not the intima. Crafoord again successfully performed this operation on a patient with coarctation of the aorta on October 31, 1944.[1] The early results in these two patients indicated that their eventual outlook should be good, for the blood pressures in the upper part of the body gradually came down to normal while the blood pressures in the lower part of the body, a few months later, rose to the normal level. On July 6, 1945, Gross[2, 3] of Boston, working independently, performed the same operation, using a slightly different technic. He employed a continuous, everting mattress suture which penetrated all layers of the aortic wall and approximated the intima to the intima.

Prior to either of these clinical successes, Blalock[4] in March, 1944, on the basis of experiments on dogs, suggested the use of the left subclavian artery to by-pass the aortic stricture. This method has since been used by Clagett,[5] Blalock and others in clinical cases when the length of the stricture is so great as to make end-to-end suture of the aortic segments impossible.

Gross has established an arterial bank,[6, 7] and has used this preserved material to repair the defect when the strictured area is unusually long. This was shown to be a feasible procedure by Carrel in 1912, but in later studies the wall of the transplanted segment "was found to be composed of connective tissue with no evidence of muscular or elastic tissues."[8] Surgical repair is now the generally accepted treatment for patients with coarctation of the aorta,[9-12] but certain important questions relative to this problem are as yet unanswered.

The arteriosclerotic changes which often occur in the aorta proximal to the atresia and the development of greatly dilated, thin-walled collateral channels, makes the procedure technically more difficult as adulthood is approached.

* Presented at the Thirteenth Congress of the International Society of Surgery on October 15, 1949 in New Orleans, Louisiana.

For this reason, and also because it is desirable to re-establish the circulation through the normal channels before irreversible vascular changes have occurred, it would seem desirable to correct the defect during childhood. Jones and Fell[12] and Johnson and Kirby[11] and others have commented on the desirability of early operation. However, these advantages might well be counterbalanced if the site of anastomosis in the immature aorta failed to increase in diameter. Should this be the result, the eventual outcome would be a stricture, relatively and actually, of serious degree. The studies herein reported were undertaken in an attempt to find the answer to this important question.

METHOD

Puppies obtained between the ages of six and eight weeks were checked for general physical fitness and for intestinal parasites. An attempt was made to correct their various disabilities and they were not operated upon until they seemed to be in normally good health.

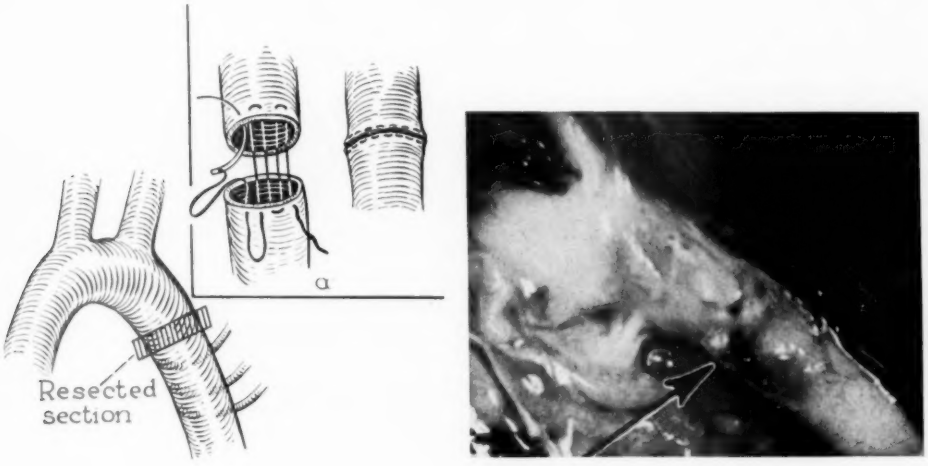
The average age at the time of operation was ten weeks, ranging from 7.5 weeks to 12 weeks. The average weight at the time of operation was ten pounds, ranging from 7.5 pounds to 16 pounds.

No preoperative medication was given. Sodium pentobarbital, in doses of 25 mg./Kg. of body weight, was given intravenously for anesthesia. An endotracheal tube was inserted in all dogs, and during the time the chest was open 100 per cent oxygen was delivered under positive pressure to maintain an adequate respiratory exchange and to saturate the hemoglobin as completely as possible with oxygen. Pure oxygen was used, because Bradshaw's work suggested that was of value in reducing spinal cord anoxia, thereby decreasing the incidence of hind quarter paralysis.[10] All animals also received intravenous plasma administered by slow drip into the dorsal metatarsal vein of the left hind leg during the operation. The rate of flow was increased as the clamps were removed from the aorta. The total amount of plasma given was 10 to 15 cc./Kg. of body weight.

The thorax was entered posterolaterally through the bed of the fifth rib, which was removed routinely. The thoracic aorta was mobilized from the left subclavian artery distally for about 4 cm. This necessitated ligation of the first two pairs, and occasionally the third pair, of aortic intercostal arteries. Care was taken to avoid injury to the phrenic and vagus nerves and to the thoracic duct.

Shortened rubber shod intestinal clamps were used to occlude the aorta. The proximal clamp was placed just distal to the origin of the left subclavian artery and the distal clamp was placed about 3 to 4 cm. below that. An average of 4.2 mm., ranging from 2 to 5.5 mm. of thoracic aorta, was resected between the clamps. The aortic segments were then anastomosed end-to-end, using 5-0 arterial silk as a continuous, everting type, mattress suture, which included all layers of the aortic wall and approximated intima to intima. The suture was started posteriorly and continued around anteriorly. The distal clamp was

first removed and the proximal clamp was then released slowly, usually requiring from two to three minutes for complete removal. The average time the aorta was occluded (from time first clamp was applied to the time the last clamp was one half removed) was 18.7 minutes, ranging from 14 to 24 minutes. At the completion of the anastomosis there was no externally visible reduction in size of the aorta at the point of union in any of the pups (Fig. 1).



A B

FIG. 1.—(A) Artist's drawing of suture method used and end result at site of aortic resection and anastomosis, and (B) photograph of dog 0011, eight days postoperative, illustrating accurate approximation of segments without stenosis, as it was done in all dogs.

Ribs four and six were then approximated with one fine wire suture and the chest wall was closed in layers with interrupted sutures of No. 30 cotton. Measurements of the inside and outside diameters of the aorta at the site of resection and anastomosis were made on each dog at the time of operation. No anti-coagulant was given these dogs either preoperatively or postoperatively. All dogs were given 200,000 units of penicillin intramuscularly each day for three days postoperatively.

All dogs were up and about their cages immediately upon reacting from the anesthesia. They were put out in runways for exercise beginning with the fourth postoperative day and have had six to eight hours of out-of-door exercise each day thereafter.

OPERATIVE RESULTS

A total of 28 pups were operated upon. Of this total, 19, or 68.0 per cent survived the operation (Table I). Of the nine, or 32 per cent, that did not survive, two died from pulmonary edema as the chest was being closed, apparently because an excessive amount of intravenous plasma had been given

TABLE I.—*Showing Pups Operated Upon; Immediate Result of Anastomosis; Postoperative Course of Each Pup; and Final Outcome of Pup.*

Dog	Anastomosis Result	Postoperative Course	Comments
01	Good. No stenosis or leakage.	Died 4 hours postoperative.	Autopsy revealed no cause of death. Anastomosis intact; no thrombus.
02	Good. No stenosis or leakage.	Died 3 hours postoperative.	Autopsy revealed no cause of death. Anastomosis intact, no thrombus.
03	Good. No stenosis or leakage.	Died 4 hours postoperative.	Autopsy revealed no cause of death. Anastomosis intact, no thrombus.
04	Good. No stenosis or leakage.	Died 3 hours postoperative.	Autopsy revealed no cause of death. Anastomosis intact, no thrombus.
05	Good. No stenosis or leakage.	Died 4 hours postoperative.	Autopsy revealed no cause of death. Anastomosis intact, no thrombus.
06	Good. No stenosis or leakage.	Died 4 hours postoperative.	Autopsy revealed no cause of death. Anastomosis intact, no thrombus.
007	Good. No stenosis or leakage.	Spastic paralysis of hind limbs. Otherwise good. Sacrificed 4-1-49.	Anastomosis intact. No thrombus. Very little stenosis.
008	Good. No stenosis or leakage.	Flaccid paralysis of hind limbs. Incontinence of bladder and rectum. Died 19th day postoperative from distemper.	Anastomosis intact. No thrombus; no stenosis; good healing.
09	Good. No stenosis or leakage.	Died 2 hours postoperative from excess I.V. fluids.	Pulmonary edema at autopsy. Anastomosis intact, no thrombus.
010	Good. No stenosis or leakage.	Died 2 hours postoperative from excess I.V. fluids.	Pulmonary edema at autopsy. Anastomosis intact, no thrombus.
0011	Good. No stenosis or leakage.	No postoperative complications from operation. Died 8th postoperative day from distemper.	Anastomsis intact at autopsy. No evidence of leakage, no thrombus.
1	Good. No stenosis or leakage.	Uncomplicated	Sacrificed 9 mo. postoperative. See RESULTS.
2	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
3	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
4	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
5	Good. No stenosis or leakage.	Slight weakness both hind legs. Otherwise, uncomplicated.	Sacrificed when full grown. See RESULTS.
6	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
7	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
8	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
9	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
10	Good. No stenosis or leakage.	Uncomplicated.	Sacrificed when full grown. See RESULTS.
11	Good. No stenosis or leakage.	Uncomplicated.	Still living. 8 mo. postoperative. To save for several years.
012	Good. No stenosis or leakage.	Died 2 hours postoperative from aspiration of vomitus.	Anastomosis intact at autopsy. No leakage or thrombus.
12	Good. No stenosis or leakage.	Uncomplicated.	Still living 8 mo. postoperative. To save for several years.
13	Good. No stenosis or leakage.	Uncomplicated.	Still living 8 mo. postoperative. To save for several years.
14	Good. No stenosis or leakage.	Uncomplicated.	Still living 8 mo. postoperative. To save for several years.
15	Good. No stenosis or leakage.	Uncomplicated.	Still living 5 mo. postoperative. To save for several years.
16	Good. No stenosis or leakage.	Uncomplicated.	Still living 5 mo. postoperative. To save for several years.

during the operation. One dog died as the result of aspiration of a large amount of vomitus about two hours postoperatively. Six died two to four hours postoperatively without reacting from the anesthetic. No obvious cause of death was found at autopsy on any one of these six dogs. In the nine dogs which failed to survive operation, the point of anastomosis was intact, with good approximation of the segments and with no evidence of leakage or thrombus formation.

One of the 19 survivors had spastic paralysis of the hind legs and developed prolapse of the rectum about three months postoperatively (Dog No. 007). She had no bladder paralysis. The aorta was obstructed for 18 minutes in this dog. She was sacrificed eight months postoperatively. The anastomosis was intact and healing was satisfactory on both gross and microscopic

TABLE II.—*Data on Survival Animals.*

Dog	Age at Operation	Present Age	Months Postop.	Weight at Operation	Present Weight	Length Aorta Resected	Time Aorta Occluded	Comments
1	11 wks.	12 mon.	9½ mon.	11 lbs.	30 lbs.	5½ mm.	22 min.	Sacrificed
2	12 wks.	12 mon.	9¼ mon.	9½ lbs.	24 lbs.	4 mm.	19 min.	Sacrificed
3	12 wks.	12 mon.	9 mon.	9½ lbs.	27 lbs.	3½ mm.	16 min.	Sacrificed
4	12 wks.	12 mon.	9 mon.	9 lbs.	26 lbs.	5 mm.	22 min.	Sacrificed
5	12 wks.	12 mon.	8¾ mon.	11 lbs.	24 lbs.	4½ mm.	23½ min.	Sacrificed
6	12 wks.	12 mon.	8¾ mon.	13 lbs.	47 lbs.	5½ mm.	24 min.	Sacrificed
7	12 wks.	12 mon.	8¾ mon.	11 lbs.	46 lbs.	4½ mm.	18 min.	Sacrificed
8	12 wks.	12 mon.	8¾ mon.	11 lbs.	34 lbs.	4 mm.	16 min.	Sacrificed
9	12 wks.	12 mon.	8¾ mon.	16 lbs.	40 lbs.	4 mm.	21 min.	Sacrificed
10	11 wks.	12 mon.	8¾ mon.	10 lbs.	34 lbs.	2 mm.	21 min.	Sacrificed
11	7½ wks.	10 mon.	8 mon.	7 lbs.	32 lbs.	3 mm.	14 min.	Living
12	9 wks.	10 mon.	7¾ mon.	9 lbs.	34 lbs.	5 mm.	19 min.	Living
13	8½ wks.	10 mon.	7¾ mon.	10 lbs.	34 lbs.	4 mm.	14 min.	Living
14	9 wks.	10 mon.	7¾ mon.	9½ lbs.	31 lbs.	4½ mm.	16½ min.	Living
15	7 wks.	5 mon.	3¼ mon.	8 lbs.	24 lbs.	4 mm.	15 min.	Living
16	7 wks.	5 mon.	3¼ mon.	6½ lbs.	15 lbs.	3½ mm.	18 min.	Living
Averages	10 wks.			10 lbs.		4.2 mm.	18.7 min.	

examination. There were no thrombi or aneurysmal dilatations but there was slight constriction of the lumen of the aorta at the anastomosis.

One of the survivors had flaccid paralysis of the hind legs with incontinence of both urine and feces (Dog No. 008). She died on the nineteenth postoperative day from distemper. The aortic anastomosis was intact, and showed no constriction or aneurysmal dilatation. There was no evidence of leakage or of thrombus formation. Healing was satisfactory on both gross and microscopic examinations.

One of the pups which survived the procedure was normal in all respects in the immediate postoperative period (Dog No. 0011), but contracted distemper and died on the eighth postoperative day. There was satisfactory healing of the anastomoses, and no constriction, aneurysmal dilatations or evidence of leakage.

Sixteen of the 19 pups which survived the procedure were in good condition postoperatively and only one showed a slight weakness in the hind legs. There was no evidence of leakage at the anastomosis or thrombus formation in any of these dogs. There were no wound infections. The findings in these 16 dogs comprise the bulk of this report.

FOLLOW-UP STUDIES AND RESULTS

All 16 were kept for study. They were maintained on adequate diets, immunized against distemper, and were put out for exercise in runways each day.

FIG. 2.—(a) Dog 10 one week postoperative and weighing ten pounds, and (b) at time of sacrifice 12 months old and weighing 34 pounds. Note no hind limb paralysis following 21-minute occlusion of thoracic aorta.

Table II shows the ages at operation and when sacrificed, and indicates the times at which studies were done. All of the dogs were full grown when sacrificed. Dogs numbered 11 through 16 had aortograms and blood pressure determinations, but were not sacrificed. They are still developing and are to be kept for long term observation and study (Figs. 2 and 3).

AORTOGRAMS

In September, 1949, aortograms were made on all 16 dogs in order that we could obtain data in regard to the relative size of the anastomosis under normal circulatory conditions. A technic, slightly modified from the one described by Robb and Steinberg.[13-15] was used. The dogs were anesthetized with sodium pentobarbital in doses of 25 mg./Kg. of body weight. They were

FIG. 3.—(a) Dog 13 one week postoperative and weighing ten pounds, and (b) at time of studies, weighing 34 pounds. This dog was not sacrificed, but will be kept for prolonged studies. Note no hind limb paralysis following 14 minute occlusion of thoracic aorta.

so placed on the table that left anterior oblique roentgenograms of the thorax could be made. This position permitted demonstration of the entire thoracic aorta with its important branches. The film-tube distance was 27 inches (MA = 150; KV = 60–65; 1/20 sec.) Twenty to 25 cc. of a 70 per cent aqueous solution of Diodrast was injected rapidly (in about one second) into the cephalic vein of the left front leg. One exposure was made five and

one-half seconds after beginning the injection. This gave excellent detail of the thoracic aorta and its important branches. All aortograms showed some constriction of the thoracic aorta at the site of the anastomosis (Figs. 4 and 5). In some there was dilatation of the thoracic aorta distal to the anastomosis.

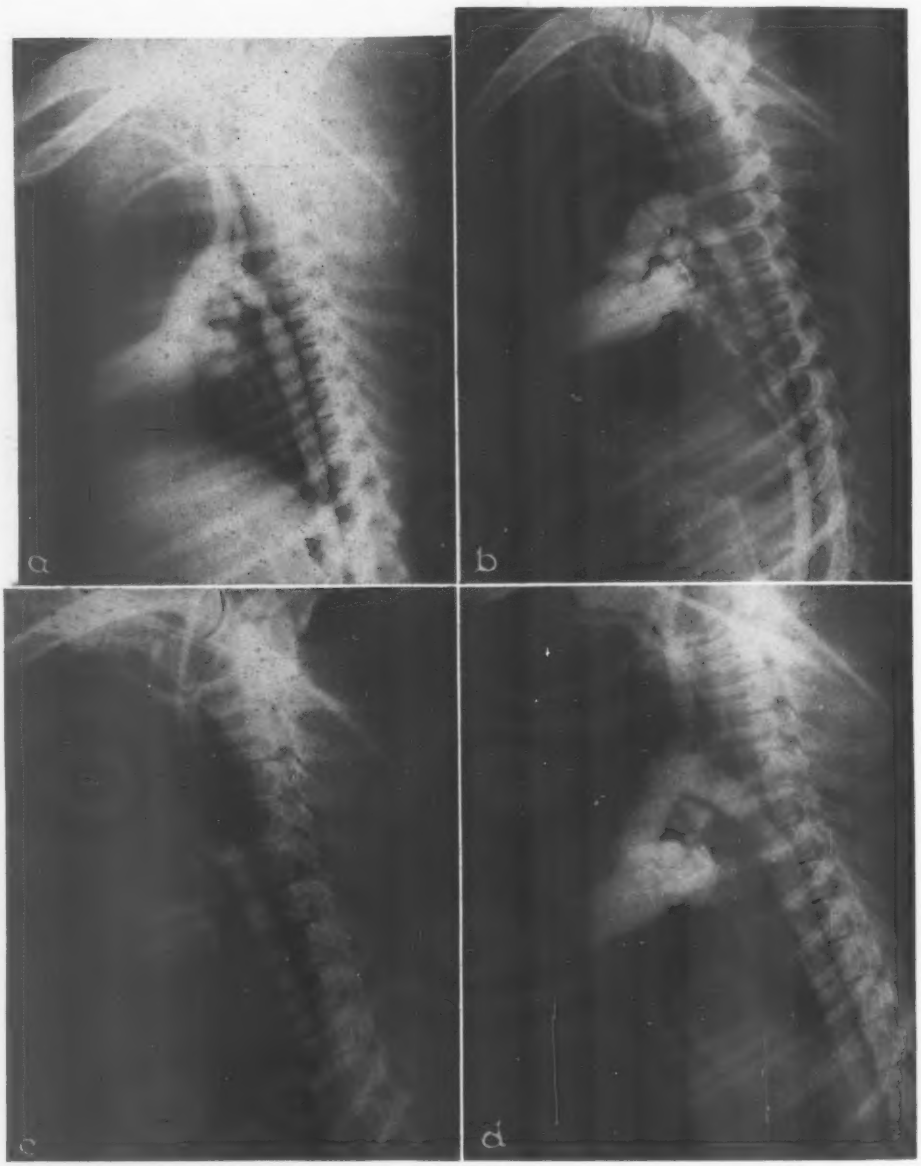


FIG. 4.—(a, b, c and d) Aortograms on Dogs 3, 5, 6, and 8, nine months post-operatively. All show some constriction at anastomosis, with no increased collateral development. All of these dogs were sacrificed.

The degree of constriction at the point of anastomosis as determined from the aortograms can be seen in Table III. The "expected normal diameter at the anastomosis" was determined by connecting normal diameters above and below the anastomosis and measuring the "expected diameter" at the point

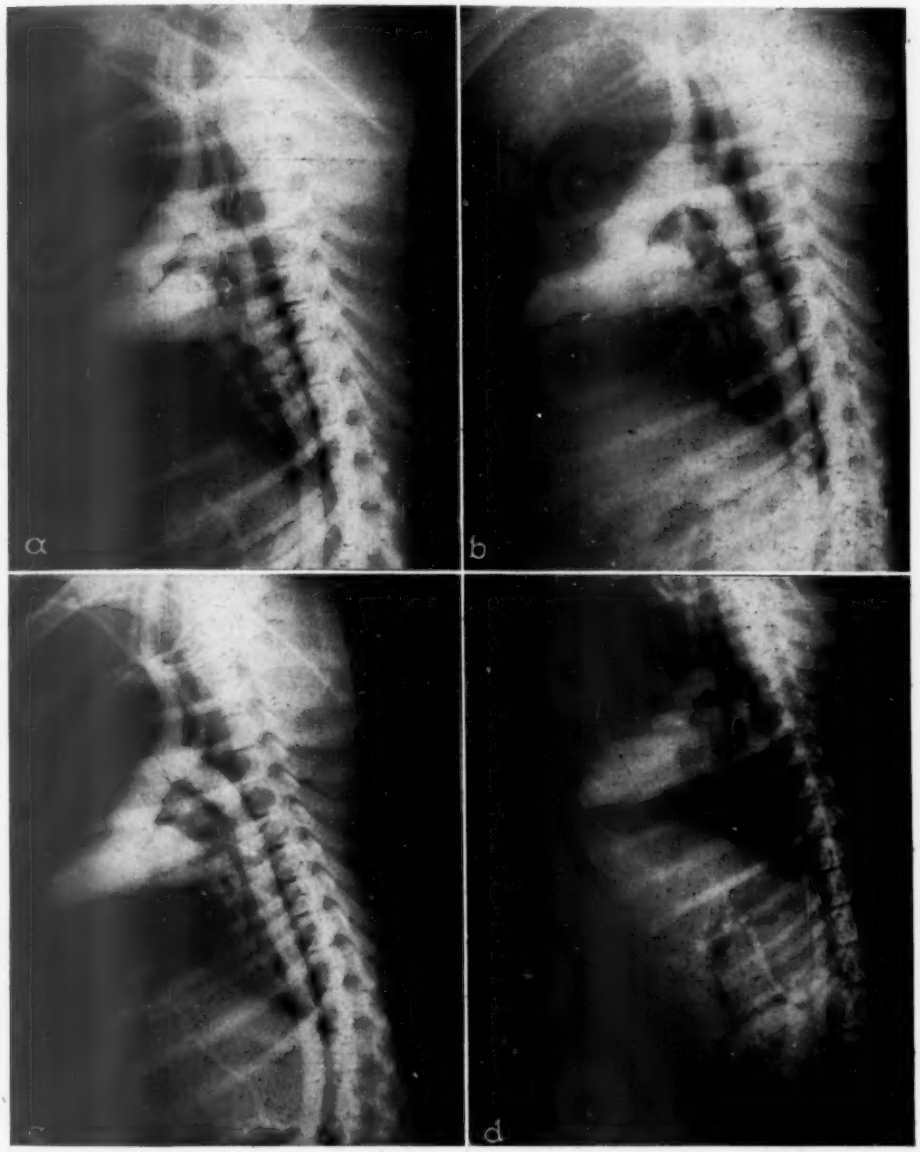


FIG. 5.—(a, b, c and d) Aortograms on Dogs 13, 14, 15 and 16, eight months, eight months, three months, and three months postoperative, respectively. None of these dogs were sacrificed.

of greatest aortic constriction. Then the diameter of the aorta at its greatest constriction at the site of anastomosis was measured (Fig. 6).

$$\frac{\text{Expected diameter in mm.} - \text{Actual diameter in mm.}}{\text{Amount stenosis in mm.}} \qquad \frac{\text{Amount stenosis in mm.}}{\text{Expected diameter in mm.}} = \%\ \text{stenosis}$$

TABLE III.—*Analysis of Measurements at Site of Anastomosis Made by Study of Aortograms on 16 Pups in Postoperative Periods Ranging From Four to Ten Months.*

Dog	Expected Normal Diameter at Anastomosis	Actual Diameter at Anastomosis	Percent Constriction at Anastomosis	Diameter of Left Subclavian Artery at Origin	Dog's Weight
1	14.0 mm.	11.5 mm.	18%	10.0 mm.	30 lbs.
2	12.0 mm.	12.0 mm.	0%	7.5 mm.	24 lbs.
3	11.0 mm.	7.5 mm.	32%	7.0 mm.	27 lbs.
4	12.0 mm.	9.0 mm.	25%	Not obtained	24 lbs.
5	11.0 mm.	9.5 mm.	14%	8.0 mm.	27 lbs.
6	13.0 mm.	10.5 mm.	20%	10.0 mm.	46 lbs.
7	14.0 mm.	11.0 mm.	21%	10.5 mm.	46 lbs.
8	15.5 mm.	12.5 mm.	20%	10.5 mm.	36 lbs.
9	14.0 mm.	11.5 mm.	18%	10.5 mm.	38 lbs.
10	13.5 mm.	10.5 mm.	22%	8.5 mm.	31 lbs.
11	14.0 mm.	10.5 mm.	25%	10.0 mm.	32 lbs.
12	12.5 mm.	10.5 mm.	16%	9.0 mm.	34 lbs.
13	12.5 mm.	10.0 mm.	20%	8.5 mm.	34 lbs.
14	14.0 mm.	10.0 mm.	29%	10.5 mm.	31 lbs.
15	11.5 mm.	8.5 mm.	26%	8.5 mm.	24 lbs.
16	8.5 mm.	8.0 mm.	6%	5.5 mm.	15 lbs.
		Anatomical stenosis (average)	20%		

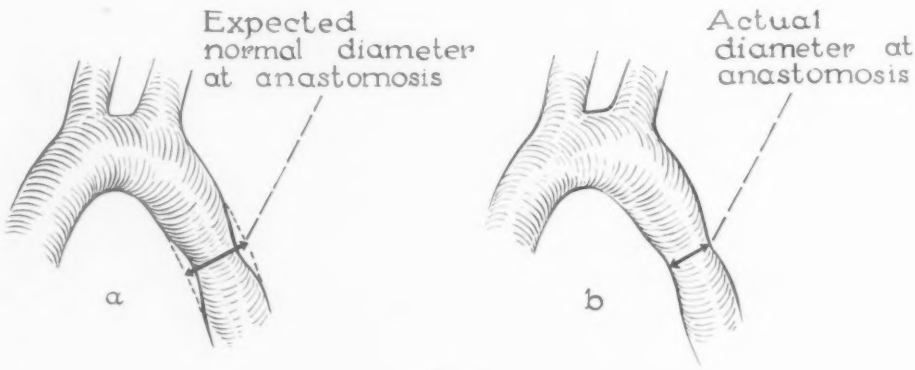


FIG. 6

There was an average of 20 per cent constriction at the anastomosis in the 16 dogs. This compares closely to that determined by direct measurement of the excised, longitudinally opened aortas. By the latter method, there was an average of 17 per cent constriction at the anastomosis. There was no evidence of increased collateral development in any of the major vessels concerned, that is the subclavian artery, brachio-cephalic artery, internal thoracic arteries, or the intercostal arteries. There did not seem to be any cardiac hypertrophy.

BLOOD PRESSURES

With the animal under sodium pentobarbital anesthesia (25 mg./Kg. of body weight given intravenously) the blood pressures were taken in the right carotid and right femoral arteries. Blood pressures were recorded 15 to 20 minutes after anesthesia with the Sylphon-Bellows manometer, utilizing an intra-arterial cannula, and also with a Lilly apparatus recording pressures through a small polythene tube. Blood pressures were elevated to hypertensive levels in all dogs, but according to Page[16] this is a constant finding with dogs anesthetized with sodium pentobarbital. A re-check by mercury manometer of the pressure readings without anesthesia supported Page's findings, as with this technic the blood pressures were normal. Pressure levels in the femoral and carotid arteries were equal, or slightly higher in the femorals in all 16 dogs, both when anesthetized and when awake.

FINDINGS AT AUTOPSY

Ten of the 16 survivors, numbers 1 through 10, were sacrificed for study during the ninth month postoperative, when they were 12 months old. The average weight of these ten dogs at the time of operation was 11 lbs., and at the time of sacrifice, 33 lbs. The remaining six dogs (11–16) are to be kept for study after more prolonged postoperative periods (Table II).

The postoperative observations in these dogs were made with especial precautions to avoid trauma, blood loss and shock. The chest was carefully opened under positive pressure so as to maintain adequate respiratory exchange. It was possible therefore to examine the area of anastomoses when the blood pressure was normal. In all animals there were light adhesions between the left upper lobe of the lung and the site of anastomosis. These were easily separated and the site of anastomosis was well exposed. There was consistent constriction of the aorta at the site of the anastomosis, but hardly so much as one would have expected after reviewing the aortograms (Figs. 7 and 8), which gave the appearance of moderate bulging of the aorta above and below the site of anastomosis. No thrill was felt at the site of, or in the vicinity of the anastomosis. There was no evidence of aneurysmal dilatation of the aortic wall at the site of the anastomosis. Aortic pulsations below the anastomosis were apparently equal in intensity to those above the anastomosis. The subclavian artery, the brachiocephalic artery, the internal thoracic arteries and intercostal arteries were exposed and observed under normal blood pressure conditions in each dog. Each of these arteries in all dogs was considered to be normal. The heart in all dogs seemed of normal size, as was the thickness of the left ventricular wall. Gross appearance of the other organs, including the kidneys and spinal cord, was normal in all dogs.

The site of aortic anastomosis, with one centimeter on either side of it, was removed and closely examined. The lumen at the site of anastomosis was found quite adequate, with no evidence of impingement upon the lumen by

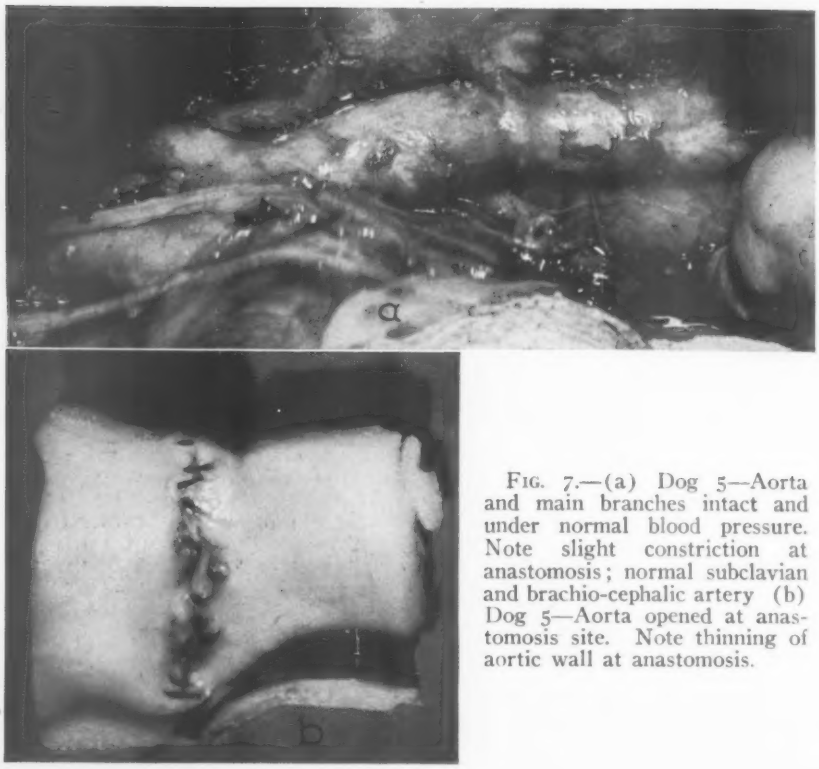


FIG. 7.—(a) Dog 5—Aorta and main branches intact and under normal blood pressure. Note slight constriction at anastomosis; normal subclavian and brachio-cephalic artery (b) Dog 5—Aorta opened at anastomosis site. Note thinning of aortic wall at anastomosis.

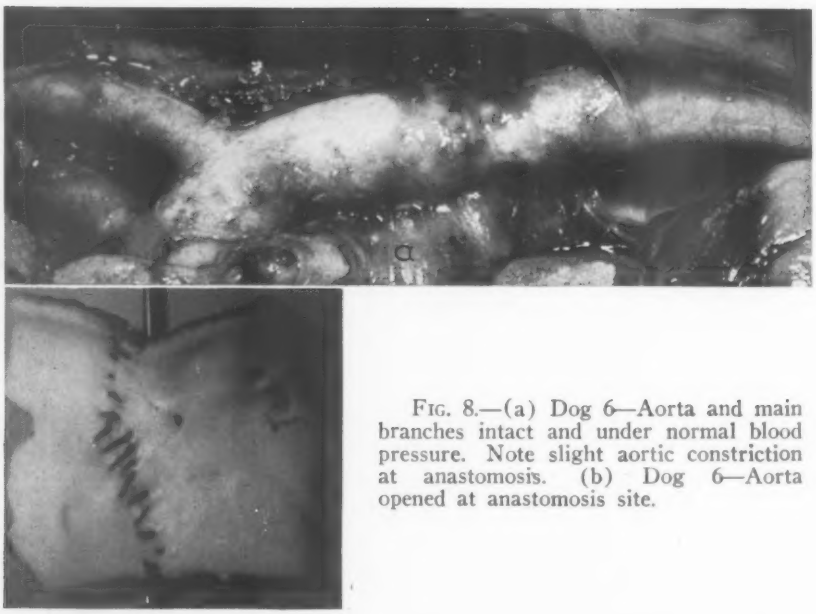


FIG. 8.—(a) Dog 6—Aorta and main branches intact and under normal blood pressure. Note slight aortic constriction at anastomosis. (b) Dog 6—Aorta opened at anastomosis site.

thrombi or folds of aortic wall (Figs. 7 and 8). The segment was then opened longitudinally and examined. The intimal surface appeared completely intact at all points and there was no evidence of thrombus formation. In some sections there was more suture material visible on the intimal surface than had

TABLE IV.—*Aortic Size.*

	Inside Diameter				Outside Diameter			
Dog	At Anastomosis at Operation	At Anastomosis at Autopsy	1 cm. Proximal to Anastomosis at Autopsy	1 cm. Distal to Anastomosis at Autopsy	At Anastomosis at Operation	At Anastomosis at Autopsy	1 cm. Proximal to Anastomosis at Autopsy	1 cm. Distal to Anastomosis at Autopsy
1	5.00 mm.	6.00 mm.	9.00 mm.	8.50 mm.	7.00 mm	7.00 mm.	11.00 mm.	9.50 mm.
2	4.50 mm.	5.60 mm.	6.50 mm.	6.50 mm.	6.50 mm	6.40 mm.	10.00 mm.	9.50 mm.
3	4.00 mm.	4.52 mm.	7.00 mm.	6.50 mm.	6.50 mm	6.20 mm.	8.50 mm.	8.00 mm.
4	4.50 mm.	6.68 mm.	8.00 mm.	6.00 mm.	7.00 mm	7.48 mm.	11.00 mm.	10.00 mm.
5	4.50 mm.	6.68 mm.	8.00 mm.	6.00 mm.	6.00 mm	7.32 mm.	11.00 mm.	8.50 mm.
6	5.00 mm.	7.32 mm.	8.50 mm.	6.50 mm.	8.00 mm	7.96 mm.	10.50 mm.	9.00 mm.
7	4.50 mm.	7.32 mm.	7.00 mm.	6.00 mm.	6.00 mm	7.96 mm.	10.00 mm.	8.00 mm.
8	5.00 mm.	7.00 mm.	8.00 mm.	7.50 mm.	7.00 mm	7.48 mm.	10.00 mm.	10.00 mm.
9	6.00 mm.	7.64 mm.	8.50 mm.	6.50 mm.	8.50 mm	8.60 mm.	11.00 mm.	9.50 mm.
10	5.00 mm.	7.32 mm.	9.00 mm.	7.00 mm.	7.00 mm	7.96 mm.	12.00 mm.	10.00 mm.
Ave.	4.80 mm.	6.70 mm.	7.95 mm.	6.70 mm.	6.95 mm	7.54 mm.	10.50 mm.	9.20 mm.
		1.90 mm. increase				0.59 mm. increase		

been expected. This we believe was due to the longitudinal stretching of the aorta, which resulted in the pulling of the continuous suture into the lumen. Looking at the cross section surface of the longitudinally cut aorta at the

TABLE V.—*Percentage Decrease in Aortic Diameter at Site of Anastomosis as Measured From Excised Aortic Segments*

Anatomical Per Cent Decrease at Anastomosis	
Dog 1 = 33%	Dog 6 = 14%
Dog 2 = 14%	Dog 7 = 0
Dog 3 = 23%	Dog 8 = 12%
Dog 4 = 15%	Dog 9 = 10%
Dog 5 = 15%	Dog 10 = 19%
Average anatomical decrease at anastomosis = 17%	

anastomosis, there was definite thinning of the media at the area of anastomosis as compared with the normal thickness proximal and distal to this point.

Table IV shows measurements of the inside and outside diameters of the aorta at the point of anastomosis at the time the operations were done. On the same charts are measurements taken at the time of sacrifice. From these it is seen that the aorta did increase in diameter at the point of anastomosis, but not to the same extent as the aorta proximal and distal to the anastomosis. The stenosis at the anastomosis from these specimen measurements was 17 per cent (Table V). That there is thinning of the wall of the aorta at the

FIG. 9

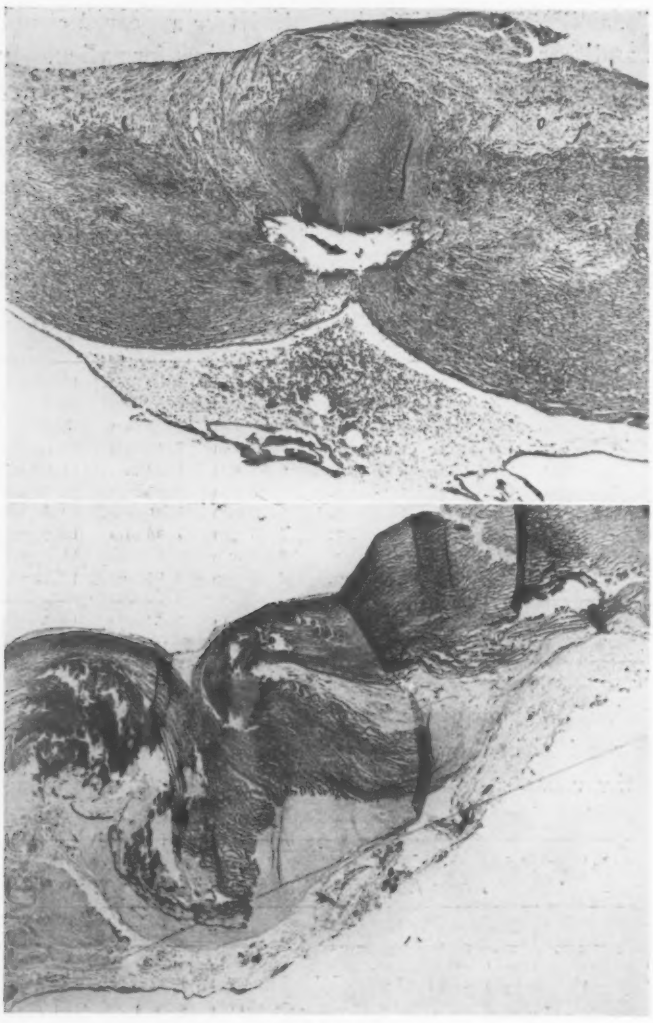


FIG. 10

FIG. 9.—Dog 0011, aorta eight days postoperative, x75. This shows the organizing exudate covered by endothelium to ensure the continuity of the intima; external to which note the site of the suture and the everted layers of the aorta which are beginning to be fused. Note at the site of the suture the compression and condensation of the elastic fibers.

FIG. 10.—Dog 9, x75; aorta Elastic Stain, nine months postoperative. This shows the healing process, with the condensation of elastic tissue at the site of the suture; eversion of the cut ends and proliferation of fibrous and elastic tissue from the cut media to fuse with the adventitial fibro-elastic tissue at a distance from the site of the anastomosis. Note that, although at the site of the anastomosis, as a result of suturing and the normal process of growth which follows as the pup grows to adulthood, the musculo-elastic layer is gradually thinned out, yet the eversion and proliferation of fibro-elastic tissue which follows makes this part of the aorta stronger and more resistant to pressure than the parts proximal and distal as observed on roentgen ray examination. This observation has been constant in all the dogs operated.

FIG. 11

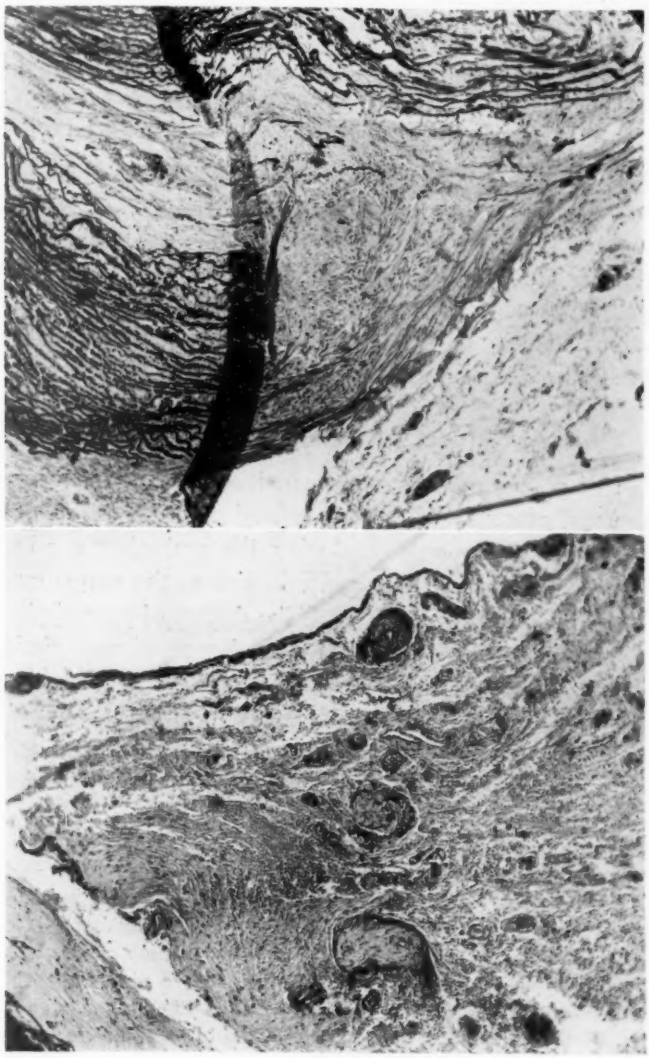


FIG. 12

FIG. 11.—Dog 9, same section x150, showing the delicate fibro-elastic fibrils connecting the severed media with the membrana elastica externa.

FIG. 12.—Aortic vessels, x75, adventitia Dog 9, close to the anastomosis, as seen in Figure 10 as well as this. The arteries and arterioles of the adventitia show marked hypertrophy of the wall and narrowing of the lumen. This process is due to a sub-intimal proliferation of fibrous tissue limited by the internal elastic lamina.

anastomosis can be seen by the measurements of the inside and outside diameters at this point (Table IV).

HISTOPATHOLOGY

Longitudinal sections were made of each aorta to include the site of anastomosis and approximately one centimeter proximal and distal to that site. Sections were taken from the anterior portion of the aortic wall and from the posterior portion of the wall from which the aortic intercostal arteries take their origin. Each section of the wall was studied carefully after staining with hematoxylin and eosin, Masson's stain, and elastic tissue stain.

The intima in all sections was healed over the area of anastomosis. In an occasional section there was a small area of endothelial proliferation over the site of a suture. This was minimal in amount and occurred infrequently. The media was well healed in all cases. The adventitia also showed good healing in all sections. The photomicrographs illustrate the essential points found in the section studies (Figs. 9 through 12).

SUMMARY

The aortas of young pups were resected and immediately anastomosed; using a continuous, everting mattress suture of 5–0 silk, which penetrated all layers of wall. Subsequent studies, during life and after sacrifice, indicate that the point of anastomosis will increase in size as the puppy grows but not as much as normal portions of the aorta. The degree of constriction was 17 to 20 per cent at the site of anastomosis. The degree of constriction was not sufficient to stimulate increased development of collateral channels or to cause cardiac enlargement. Healing at the point of anastomosis was excellent and adequate to withstand normal aortic pressures. Six of the dogs are being kept for study over a longer period in an attempt to determine the final result of this surgical procedure.

The author wishes to acknowledge the valuable assistance of Vina I. Brooks, R.N. during the period of surgery of the pups; the support of Col. W. H. Moursund under whose command this work was done; the advice and co-operation of Dr. I. A. Bigger and Dr. E. I. Evans, in whose department the final studies were made; the aid of Dr. Ken Carter, Department of Roentgenology, Medical College of Virginia; and the valuable assistance of Dr. Philip F. Sahyoun in the study of the histopathologic sections.

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RECURRENT DISLOCATION OF THE SHOULDER JOINT*

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A SURVEY OF THE LITERATURE on the pathogenesis of recurrent dislocation of the shoulder reveals that there is total lack of agreement on the causative factors responsible for this lesion. It is interesting to note that most investigators, when considering the pathology of this disease, fail to evaluate the glenohumeral joint in its entirety but rather restrict themselves to some local irregularity observed in the labrum glenoidale, fibrous capsule or head of the humerus. Another observation that is outstanding by its very inconsistency is that these same workers believe and teach that stability of the glenohumeral joint is directly dependent upon the surrounding intricate muscular apparatus that motivates it; yet, when a state of instability exists, local defects in other components of the joint are credited as causative agents.

Bankart stoutly defends his belief that the pathology in all instances is either a detachment of the labrum from the anterior glenoid brim or a tearing away of the capsule from the labrum. He goes so far as to express the view that the recurrent lesion is a different entity from the ordinary acute traumatic dislocation. The recurrent lesion, according to his view, is invariably an anterior dislocation which tends to recur because the fibrocartilaginous labrum fails to re-attach itself to the glenoid margin. The acute traumatic lesion is an inferior dislocation, the head being forced through a rent in the fibrous capsule which heals readily to bone, thereby preventing recurrences. These features of the morbid anatomy of recurrent dislocation were recorded by Broca and Hartman as far back as 1890. These observers also noted a defect on the posterior aspect of the humeral head which they believed facilitated intracapsular subluxation of the head of the humerus.

Some observers are convinced that defects in the humeral head are capable of producing dislocation and believe that the frequency of the dislocation depends upon the size of the humeral defect. As the defect increases in size, the tendency to dislocation becomes greater.

The great discrepancy in the etiologic factors responsible for the lesion, and the lack of uniformity in the surgical principles employed to effect a cure, prompted this investigation. Observations noted herein lead one to conclude that the local pathologic abnormalities recorded above are not the true causative agents, and that many of the surgical procedures performed to cure this malady are based on erroneous interpretations of normal or variational anatomy. Moreover, this investigation provides an explanation for the numerous failures which occur in procedures utilizing the principle of suspension to effect a cure,

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and for the success of those procedures which shorten the structures on the anterior aspect of the glenohumeral joint. The conclusions arrived at in this investigation are based on: (1) a study made on 36 recurrent dislocating shoulders. These shoulder joints were thoroughly explored and all abnormalities of the inside of the glenohumeral joint recorded; (2) gross and microscopic observations noted in 88 shoulder joints explored postmortem of 44 individuals ranging in age from 18 to 79 years who, prior to their death, gave negative histories and showed no clinical evidence of dysfunction of the shoulders; (3) determination of the range of external rotation of the arms in 800 normal individuals, 100 for each decade, from the first to the eighth inclusive; and (4) a postoperative end-result survey of 23 recurrent dislocating shoulders treated by the Magnuson (modified) procedure, the shortest interval since operation being 17 months, the longest three and a half years.

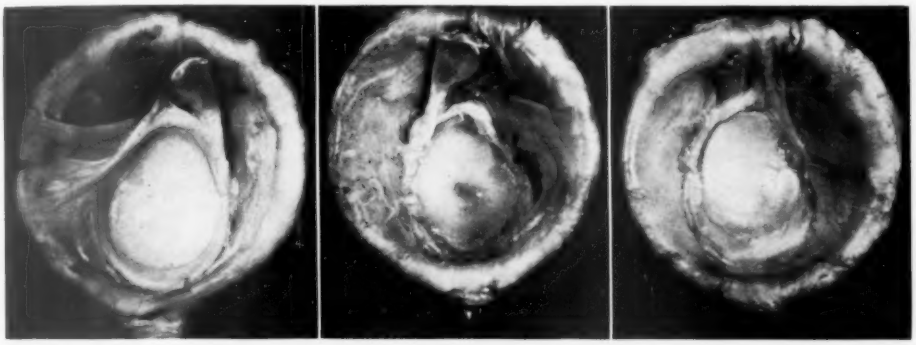


FIG. 1 FIG. 2 FIG. 3

FIG. 1.—Specimen exhibits one large subscapularis recess immediately above the middle glenohumeral ligament. Superior, middle and inferior glenohumeral ligaments are well defined. Note the intimate relationship of the biceps tendon and the glenohumeral ligament as they blend with the labrum glenoidale. There is early recession of the labrum from the brim of the upper half of the glenoid cavity.

FIG. 2.—Two subscapularis recesses exist; one above and one below the glenohumeral ligament. Note the severe degenerative changes that are apparent in the entire labrum glenoidale; these changes comprise shredding, thinning and villous formation on all surfaces of the fibrocartilaginous structure. Figures 1 and 2 comprise 88.6 per cent of all specimens studied.

FIG. 3.—No subscapularis recesses are demonstrable in this specimen. This pattern of the inside of the glenohumeral joint comprises 11.4 per cent of the specimens studied. Note the definite separation of the labrum from the upper half of the brim of the glenoid cavity.

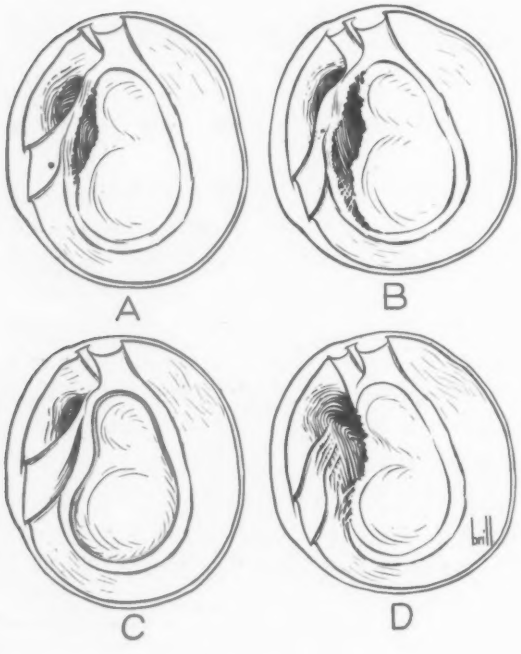
ANATOMY OF THE INSIDE OF THE GLENOHUMERAL JOINT

At this point a review of the variational anatomy of the inside of the glenohumeral joint is imperative. In a study conducted by Dr. G. Bennett, Dr. G. Callery and the author* on the variational anatomy and degenerative lesions of the shoulder joint, it was noted that the fibrous capsule in the anterior por-

* All figures in this article dealing with the variational and gross degenerative changes of the inside of the glenohumeral joint were taken from this study which appeared in the American Academy of Orthopedic Surgeons Instructional Course Lectures, Vol. VI, 1949.

FIG. 4

FIG. 5



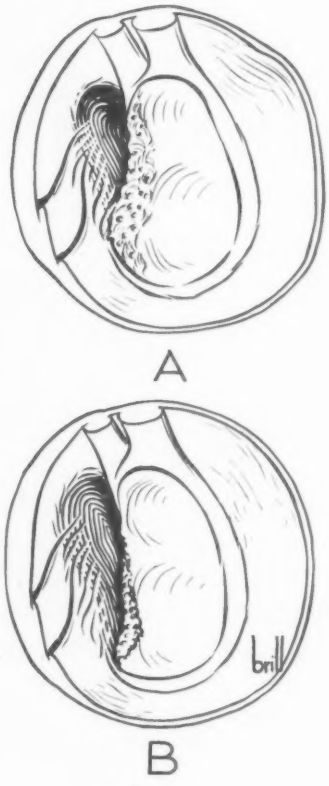


FIG. 6

FIG. 7

(Legend on opposite page.)

tion of the joint is not, as so many believe, continuous with the capsular surface of the labrum glenoidale. Instead it is projected mesially as far as the subcoracoid region and then reflected onto the anterior surface of the neck of the scapula until it reaches the periphery of the labrum. This out-pouching of the capsule forms the subscapularis recesses, one or two, depending on the variable middle glenohumeral ligament. In joints possessing one bursal recess it is usually located below the middle glenohumeral ligament; those possessing two disclose one recess above and one below the ligament. These two patterns comprised 88.6 per cent of all the specimens studied (96 shoulder joints). In the remaining 11.4 per cent there were no recesses demonstrable; the capsule in these cases was continuous with labrum (Figs. 1, 2 and 3). Although there is pronounced variation in the size of the subscapularis recesses, in general they are rather spacious. It is conceivable that one not familiar with this variational anatomy might readily misinterpret these recesses as tears or rents in the capsule. The subscapularis muscle and tendon are in intimate relationship to the anterior surface of the bursal recesses.

It becomes apparent that it is impossible for the head to pierce the capsule in the anterior portion of the joint unless it is forced through a rent below the subscapularis muscle. The head, therefore, in recurrent dislocations lies within the subscapularis recess or recesses, which are stretched to accommodate it. Both the middle and inferior glenohumeral ligaments may be the only feeble barriers to dislocation.

PATHOLOGIC OBSERVATIONS NOTED AT OPERATION ON 36 RECURRENT DISLOCATING SHOULDERS

Thirty-six individuals provided the 36 recurrent dislocating shoulders studied; there were 35 males and one female; the age ranged from 18 to 36 years.

Musculotendinous Cuff. An observation of considerable significance was the pronounced laxity of the musculotendinous cuff in all cases. It appeared as if the short rotators were unduly stretched and lacked normal tonicity. Mild traction in all instances, with the cuff intact, readily separated the articulating surfaces of the humeral head and glenoid cavity. Such laxity in the

FIG. 4.—The type of complete tear noted in two of the 36 recurrent dislocating shoulders explored.

FIG. 5.—The type of tear noted in the subscapularis region of the musculotendinous cuff in three of the 36 shoulders explored.

FIG. 6.—(a) Only moderate detachment of the labrum. (b) Severe detachment of the labrum from the entire anterior surface of the glenoid cavity. (c) Slight elevation from its bony attachment of glenoid border of the fibrocartilaginous structure, resembling in a large measure the menisci of the knee joint. This type of separation was found in 7 cases or 19.4 per cent of shoulders explored. (d) Detachment and complete degeneration of the anterior portion of the labrum and also fraying and some shredding of the middle glenohumeral ligament.

FIG. 7.—(a) Complete degeneration of the labrum is noted anteriorly with some involvement of the middle glenohumeral ligament and also degenerative bone changes along the anterior margin of the glenoid cavity. (b) Note the defect on the anteroinferior aspect of the glenoid cavity resulting from actual loss of bone substance, as if a piece of bone had been sheared away from this region.

musculotendinous cuff is not demonstrable in normal shoulders even under deep anesthesia.

In two instances small tears not over one centimeter wide were demonstrable in the supraspinatus region of the cuff (Fig. 4). The margins of the defects were smooth, indicating that the lesions were not recent. Three shoulders disclosed partial detachment of the inferior portion of the subscapularis tendon at its insertion into the lesser tuberosity (Fig. 5). Upon division of the subscapularis tendon at its insertion into the anterior lip of the bicipital groove, the humeral head could be readily dislocated anteriorly by external rotation of the extremity.

Labrum Glenoidale and Fibrous Capsule. Some degree of labral detachment was discernible in 29 cases (80.5 per cent). The detachment was invariably from the anterior or antero-inferior portion of the glenoid rim. All degrees of detachment were noted, varying from one or two centimeters in length to complete detachment of the entire anterior one-half of the fibrocartilaginous ring (Fig. 6). In most instances the capsule and periosteum together with the labrum were stripped for varying distances from the anterior surface of the neck of the scapula. This last feature was more pronounced in cases with extensive labral detachments. Such a defect comprises the classic "Bankartian Lesion." Seven cases (19.4 per cent) disclosed the labrum to be firmly anchored to the glenoid margin by its capsular border, its glenoid border, however, being free like a meniscus (Fig. 6c). Many of the detached labra disclosed advanced fraying, shredding, and thinning.

Glenohumeral Ligaments and Subscapularis Recesses. In many instances the middle ligament could not be identified. In a few, small shreds of tissue indicated the remains of the ligament (Fig. 7). In most shoulders, the inferior ligament was discernible but it was greatly attenuated and stretched. All shoulders revealed marked stretching of the subscapularis recesses, and one could pass a probe mesially on the anterior surface of the neck of the scapula as far back as the coracoid process.

Bone Changes. Varying degrees of erosion and eburnation were found on the anterior lip of the glenoid fossa in cases in which there were labral detachments. In one instance a large irregular defect was demonstrable, leading to the conclusion that a fragment of bone had been sheared away from this area (Fig. 7b). Some osteophytes were observed on the anterior surface of the neck in three cases with extensive labral detachment.

PATHOLOGIC OBSERVATIONS NOTED IN THE MUSCULOTENDINOUS CUFF OF 44 NORMAL SHOULDER JOINTS

It was interesting to note that the alterations noted above in the labrum glenoidale, fibrous capsule, and glenoid cavity did not essentially differ from the degenerative lesions observed in normal shoulder joints. Also, the lesions found in the normal shoulders were identically the same, in the respective decades, as those discernible in the aforementioned investigation, Variational Anatomy and Degenerative Lesions of the Shoulder Joint, a study conducted

on cadavers on which no medical histories or physical examination prior to death were available.

In infant shoulders the labrum blends with the hyaline cartilage of the humeral head; so complete is the fusion of these two structures at this period of life that, microscopically, they appear as one structure (Fig. 8). As early as the second decade, evidence of tearing away of the triangular fibrocartilage is manifested (Fig. 9a and b). This gradual separation is noted grossly in the second decade and increases in frequency and severity in each subsequent decennium; after the sixth decade it is demonstrable to some degree in approximately 100 per cent of the cases (Fig. 10a and b). In the light of this information it becomes apparent that labral detachment is associated with advancing age. The cause for labral detachment is found in the topographical relation of the biceps tendon and glenohumeral ligaments to the labrum.

FIG. 8.—Microscopic section through the junction of the labrum and the glenoid cavity of a child one year of age. Note the intimate blending of the fibrocartilage on the left with the hyaline cartilage of the glenoid cavity on the right. No definite line of demarcation exists. Both the fibrocartilage and the hyaline cartilage appear to fuse into one structure.

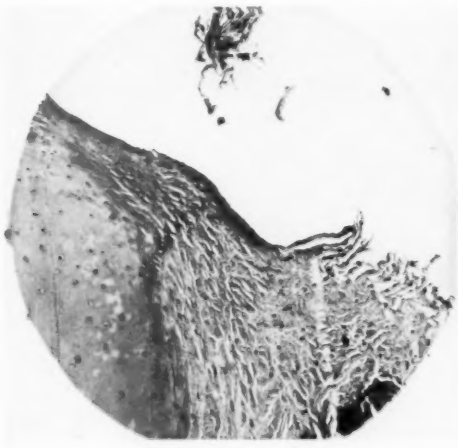


FIG. 9A

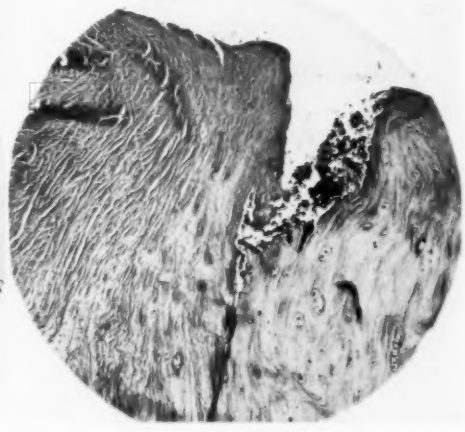
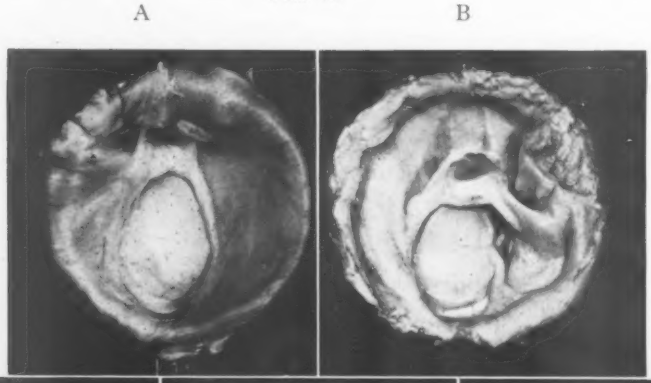


FIG. 9B

FIG. 9.—(a) Specimen through the junction of the cartilage and the labrum from an individual in the second decade of life. Note the gradual tearing away of the labrum from its attachment to the hyaline cartilage. (b) Specimen in the third decade of life. Note the actual splitting away of the fibrocartilage on the left from the glenoid cavity on the right.

Both the biceps tendon and the glenohumeral ligaments blend with the fibers of the labrum; any traction on these structures will tend to tear away the labrum from its bony anchorage on the glenoid (Fig. 1). Comprehension of this mechanism is facilitated by the knowledge that the glenohumeral ligaments act as check reins to external rotation of the limb. It becomes apparent that during normal joint function distracting forces are acting constantly on the

FIG. 10



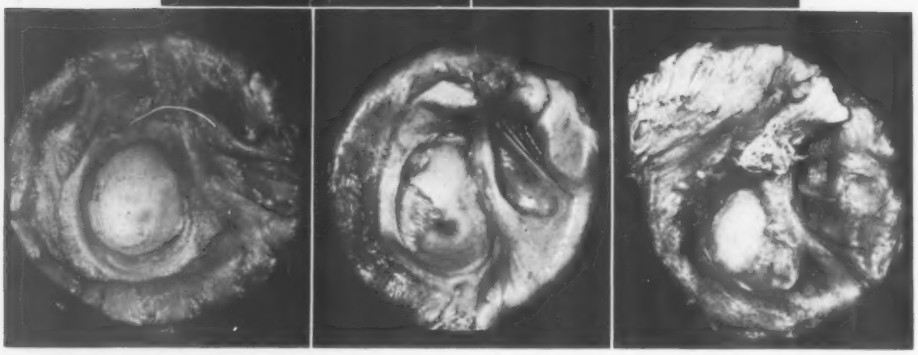


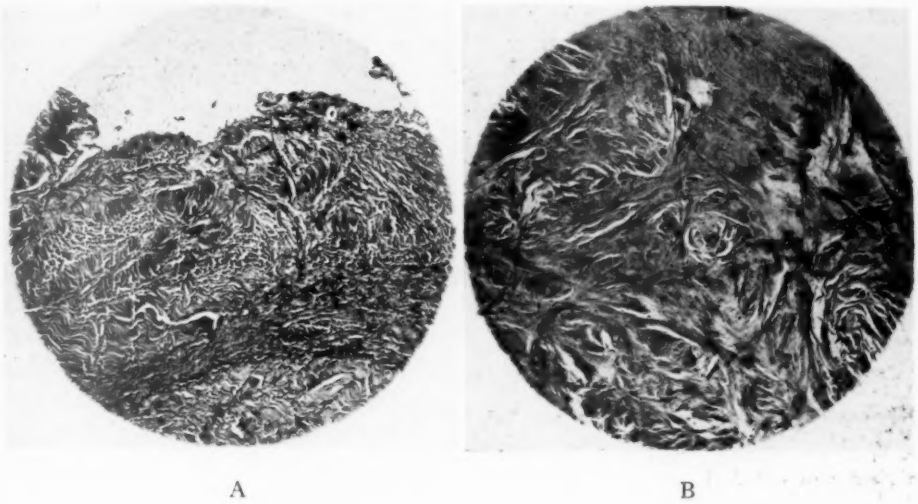
FIG. 11

FIG. 10.—(a) A specimen from the second decade in life. Note the separation of the labrum from the glenoid cavity along its upper borders. (b) Note the complete separation of the labrum from the entire brim of the glenoid cavity. The only ligament that is firmly attached to the glenoid cavity is the inferior glenohumeral ligament.

FIG. 11.—(a) There is marked thickening of all the soft tissue structures comprising the inside of the glenohumeral joint. Note the marked thickening of the labrum, middle glenohumeral ligament, the inferior and superior glenohumeral ligament, and all the synovial tissues lining the joint. It appears as if the hyperplastic process is gradually obliterating the subscapularis recess in the upper right hand corner. (b) Again there is marked thickening of all the structures of the inside of the joint. There is detachment of the labrum from the upper half of the glenoid cavity, also an attempt is made by nature to re-attach the labrum to its original bony site by the formation of villi and tabs between the labrum and the glenoid cavity. This is particularly noted in the posterior aspect of the joint. (c) There is profound thickening of the synovial and sub-synovial tissues, the fibrous capsule, the labrum glenoidale, and all the glenohumeral ligaments. The process is so marked that it is hardly possible to distinguish the different glenohumeral ligaments. Also note marked degenerative changes in the form of villi, shredding and thickening of the biceps tendon.

labrum through the medium of the biceps tendon and glenohumeral ligaments; these forces achieve, gradually, separation of this triangular fibrocartilage from its bony attachment.

Grossly, another observation of great significance was the gradual but progressive increase in degenerative changes in each subsequent decade after the third in the synovialis, fibrous capsule, glenohumeral ligaments, musculotendinous cuff, and biceps tendon. The lesions comprised tearing, shredding, fraying, and villous formation of the synovialis; tearing and shredding of the cuff, particularly in the suprespinatus and infraspinatus areas; and a diffuse thickening of all the above components of the joint (Fig. 11a, b and c). Microscopic study disclosed a generalized increase in fibrous tissue in all the aforementioned structures. In some instances the fibrosis, especially in the



A B

C

FIG. 12.—(A) Microscopic section through one of the glenohumeral ligaments. Note the thickening of the entire synovial tissues which has been entirely replaced by fibrous tissue. (B) This represents a section through the synovial membrane of an individual in the fifth decade of life. Note the marked fibrosis present and hyalinization of the fibrous tissue. (C) Microscopic section through the musculotendinous cup in the region of the supraspinatus tendon. Note the advanced degenerative process characterized by fragmentation, shredding, tearing, of all the tendon fibers and re-placement of the degenerated tissue by fibrous tissue.

later decades, was so pronounced that it partially obliterated the bursal recesses (Fig. 12a, b and c). It was also interesting to note that in the late decades of life (especially after the sixth decade) the hyperplastic process was exceedingly pronounced; it appeared as if nature were attempting to re-attach detached labra to the glenoid brim. In many instances the attempt was in part successful. It is logical to assume that replacement of normal elastic tissue by fibrous tissue must result in varying restriction of motion of the joint depending upon the severity of the process of fibrosis. This destruction led to the next step in the investigation.

DETERMINATION OF THE RANGE OF EXTERNAL ROTATION IN 800 NORMAL INDIVIDUALS

Eight hundred individuals, 100 in each decade from the first to the eighth inclusive, were studied. All possessed normal shoulders. No individual with a history of shoulder disability or who on examination revealed clinical evidence of some pathologic disorder referable to the shoulder joints was included in this survey. There was found an average range of external rotation in the second decade of 105 degrees, in the third of 85 degrees, in the fifth of 78 degrees, and in the seventh and eighth of 68 degrees (Fig. 13). It becomes obvious that the above increase in fibrosis which normally occurs with advancing age is responsible for a gradual decrease in the range of external rotation in each successive decade.

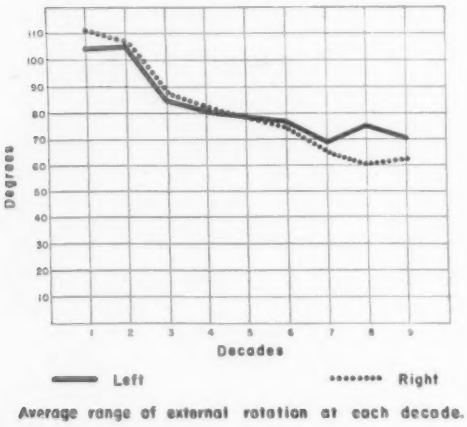


FIG. 13.—Graph showing the gradual restriction of external rotation from the first to the eighth decades of life.

CORRELATION OF OBSERVATIONS AND CLINICAL FACTS

Progressive fibrosis of all capsular tissues, followed by restriction of external rotation, provides an explanation for the following clinical facts, namely, that recurrent dislocation of the shoulder is seldom encountered after the fourth decade and that the malady is a self-limiting disease. Many instances are known of individuals who had in early life numerous recurrences which, without treatment, steadily decreased in frequency as the individual approached middle life; finally no more occurred. In other words, nature's method of curing the disease is by producing scarring and fibrosis of the soft tissues sufficient to limit markedly external rotation.

Another observation of significance is that in those decades in which labral detachments are most frequent and most severe (after the fourth decade) recurrent dislocations are rarely encountered. In the light of these observations one is forced to conclude that local lesions such as labral defects and tears in the capsule and defects in the humeral head are not the causative agents of the

disease, that a true Bankartian lesion does not exist, and that some other disorder is the responsible agent.

CONCEPT OF PATHOGENESIS OF RECURRENT DISLOCATION OF THE SHOULDER

Capsular, labral lesions and humeral head defects are not the prime causative agents of recurrent dislocation; they are changes associated with ageing but may be produced or aggravated by trauma. Neuromuscular imbalance, chiefly of the short rotator muscles, is the most single important causative factor. Such a neuromuscular state follows severe stretching of and direct injury to the involved muscles, particularly the subscapularis muscle, at the time of injury. Pronounced capsular stretching and enlargement of the bursal recesses to accommodate the head are secondary adaptive changes. Repeated dislocations increase the neuromuscular imbalance, hence increasing the tendency to recurrences until nature overcomes the laxity of all tissues by a progressive process of fibrosis which limits external rotation and stabilizes the glenohumeral joint.

If the aforementioned concept of the pathogenesis of recurrent dislocation is accepted, it becomes apparent that such a complication may follow any initial mechanism of anterior dislocation, provided the short rotators, particularly the subscapularis muscle, are severely stretched and traumatized, and if the primary dislocation has not been adequately treated. I am in total disagreement with Bankart's postulate that recurrent dislocation is a different lesion than ordinary acute traumatic dislocation and that it can occur only in the presence of capsular or labral detachment.

Capsular and labral lesions, as well as defects in the humeral head, have been given an undeserved place of importance in the pathology of recurrent dislocation, because it has been clearly revealed in this study that they are normal degenerative changes associated with wear and tear and senescence—although it must be admitted that trauma plays a part in their formation.

ANALYSIS OF OPERATIVE PROCEDURES

A survey of the many operative procedures devised for re-dislocation reveals that the essential feature which affects a cure is restriction of external rotation. Regardless at what region of the glenohumeral joint, or at what components of the joint the operative attacks are aimed, limitation of external rotation will effect a cure in the great majority of cases. This is also nature's method of eliminating the disability. Therefore, the simplest procedure which will bring about restriction of the arc of external rotation will eventually be uniformly adopted.

Magnuson points out that by the transference of the subscapularis tendon to the greater tuberosity a musculotendinous sling or cup is formed around the humeral head in both external and internal rotation which counter-balances the powerful pull of the adductor muscles (pectoralis major, latissimus dorsi, and teres major) which tend to force the head downward and forward. We are of the opinion that the Magnuson procedure properly performed will supplant all other operations.

The Magnuson procedure was modified in order to increase the efficiency of the musculotendinous sling. This has been accomplished by transferring the subscapularis tendon across the bicipital groove and anchoring it at a lower level than its original insertion onto the humeral shaft.

Magnuson Operation (Modified). (Fig. 14) An "S"-shaped skin incision, beginning at the inferior margin of the acromioclavicular joint, is made on the anterior aspect of the shoulder. The interval between the deltoid and the pectoralis major is developed, taking care not to injure the cephalic vein which is retracted medially with the pectoralis major muscle. By external rotation of the shaft of the humerus, the subscapularis tendon, as it inserts into the lesser tuberosity, comes into view. A blunt dissector is passed under the subscapularis tendon in order to determine more clearly its upper and lower

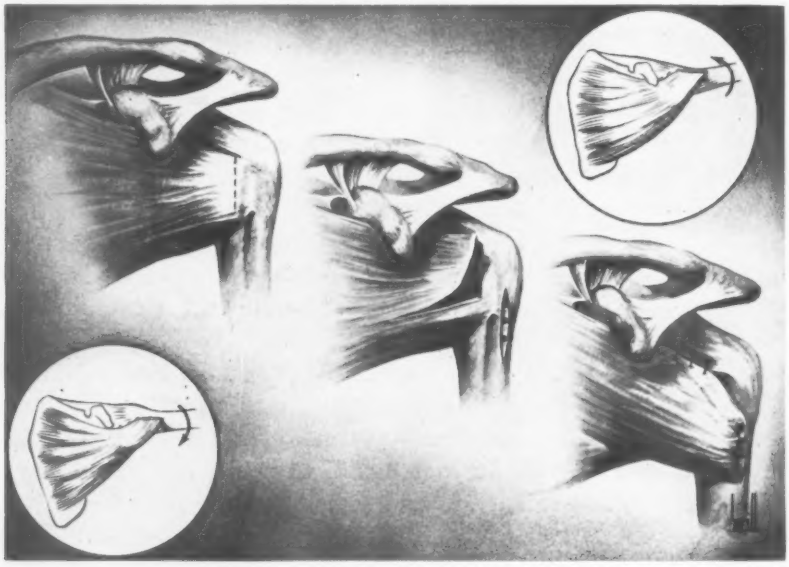


FIG. 14.—Note that in the modification of the above Magnuson operation the subscapularis tendon is anchored in a bony trough below the level of its original site on the other side of the bicipital sulcus.

borders. An incision is made in the interval between the supraspinatus and subscapularis muscles, beginning proximal to the blending of the subscapularis tendon with the fibrous capsule; the incision is continued to the anterior lip of the bicipital sulcus. A second incision, the same length as the first, is made along the lower border of the scapularis muscle. The tendon between the two incisions is then freed from the anterior lip of the bicipital groove by sharp dissection. Retraction medially of the tendon and capsule affords a clear view of the humeral head, anterior glenoid margin, and the anterior portion of the synovial capsule with its glenohumeral ligaments and bursal recesses.

The greater tuberosity is visualized by internal rotation of the arm. The subscapularis is then pulled, by means of a suture through its substance, across

the bicipital groove to a point below the greater tuberosity and its site of re-attachment is determined. The tendon should be anchored to the humeral shaft below the level of its original insertion, under moderate but not severe tension. With a thin osteotome a slot one-quarter inch wide and as long as the width of the subscapularis tendon is made parallel to the posterior lip of the bicipital groove below the greater tuberosity. Four drill holes are then made in the posterior lip of the newly formed slot and the end of the tendon is buried in the bony trough with silk mattress sutures. The upper border of the subscapularis muscle is approximated to the supraspinatus muscle by side-to-side sutures, while its lower border is sutured to the capsular tissues under the head of the humerus.

Considerable restriction of external rotation is demonstrable at the completion of the procedure. The subscapularis muscle and tendon fibers can also be seen to form a sling under the head, on abduction of the arm in internal and external rotation. The procedure is completed by wound closure in layers with interrupted sutures.

POSTOPERATIVE TREATMENT

For the first two weeks, the arm is fixed to the side with the forearm across the chest by a Velpeau dressing. For the next two weeks the arm is kept in a sling. Motion is then begun but not permitted above the horizontal plane for two more weeks. Forceful external rotation during this period is prohibited, but full resumption of all motions should be attained by the eighth week. As a rule, abduction is restricted a few degrees and external rotation may be restricted as much as 50 per cent. This, however, produces no functional disability and is assurance against re-dislocation. The amount of external rotation lost is but a small price to pay for the cure of such a disabling malady by so simple a surgical procedure.

Analysis by many observers of the different types of operations performed discloses that the Bankart and Putti-Platt procedures give a high percentage of cures while the Nicola and Henderson operations result in a high percentage of failures. Adams recorded the following data in 111 cases under observation not less than two years from the time of operation. Re-dislocation occurred in 21 (36 per cent) of 59 cases, in which the Nicola operation was employed; in two of 37 cases in which the Putti-Platt operation was done and in one of 18 cases in which the Bankart procedure was performed. Leeds reported recurrences of dislocation in nine out of 13 cases treated by the Henderson operation. Re-dislocation occurred in all cases in which the sling was constructed of fascia lata and in four of seven cases in which the tendon of the peroneus longus muscle was used as a sling.

In this investigation 23 recurrent dislocating shoulders were treated by the modified Magnuson Operation; there were recurrences of dislocation in two out of 23 cases (8.7 per cent) (Table I).

Success of the modified Magnuson and Bankart procedures can be directly attributed to the known restriction of external rotation which is achieved by

these operations. Failure of the Henderson operation can be explained on the basis that it fails to limit external rotation while the high percentage of failures occurring in the Nicola procedure results from failure to restrict external rotation and pulling away of the labrum from the glenoid rim when the weight of the extremity acts upon it through the medium of the proximal end of the biceps tendon which now functions as a suspensory ligament.

TABLE I.—*Analysis of 23 Magnuson Procedures (Modified).*

Name	Sex	Age	Date Operation	Number Recurrences	Pain	Satisfied
C.F.	M	28	5/26/48	1	Yes	No
R.M.	M	23	11/22/47	0	No	Yes
R.C	M	30	5/30/48	0	No	Yes
G.H.	M	24	10/3/47	0	Occasionally	Yes
W.M.	M	24	6/28/48	0	No	Yes
A.A.	M	23	2/30/48	0	No	Yes
M.K.	M	21	3/21/48	0	No	Yes
E.H.	M	22	4/29/48	0	No	Yes
J.C.	M	29	6/25/48	0	No	Yes
J.F.	M	26	7/30/48	0	No	Yes
W.V.	M	20	7/19/48	0	Yes	Yes
A.V.	M	25	8/24/48	6	After dislocation	No
W.S.	M	21	3/3/48	0	No	Yes
J.N.	M	18	11/15/47	0	No	Yes
T.J.	M	36	8/4/48	0	No	Yes
D.L.	M	17	5/21/45	0	No	Yes
W.B.	M	30	1/21/48	0	No	Yes
C.M.	F	29	3/26/48	0	Slight	Yes
J.C.	M	19	8/24/47	0	No	Yes
L.B.	M	33	6/12/47	0	No	Yes
T.N.	M	22	8/18/46	0	No	Yes
D.O.	M	29	10/15/47	0	No	Yes
J.N.	M	22	2/23/48	0	No	Yes

Analysis of Cases:

Total number of cases	23
Age range	17 to 33 years
Males	22
Females	1
Recurrences of dislocation	2 cases (8.7 per cent)
Pain present in	2 cases
Not satisfied with operation	2
Satisfied with operation	21
Period of observation	17 months to 3½ years

CONCLUSION

1. Local lesions of the labrum glenoidale, fibrous capsule, glenoid cavity, and humeral head are not the causative agents responsible for recurrent dislocation of the shoulder.

2. These lesions are observed in normal shoulders and are manifestations of wear, tear, and senescence.

3. Trauma does play a role in their formation.

4. They occur most frequently and exhibit the greatest severity in decades in which recurrent dislocations are seldom encountered.

5. Recurrent dislocation of the shoulder is a self-limiting disease; nature affects a cure by a progressive process of fibrosis which restricts external rotation.

6. Operative procedures which mimic this mechanism are favored by a high percentage of cures; those that do not, result in a high percentage of failures.

7. The modified Magnuson operation is offered as a simple and effective method to achieve the desired restriction of external rotation.

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EXPERIMENTAL ANASTOMOSES OF THE PANCREATIC DUCT*

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RESECTION OF THE HEAD OF THE PANCREAS requires some disposition of the remaining gland and its duct. In early experiences with this operation, it was considered too hazardous and time-consuming to attempt maintenance of the external pancreatic secretion, and the duct was therefore tied off and the end of the gland oversewn. External pancreatic fistula was a common sequel of this procedure, and impaired digestion was troublesome in a smaller number of patients. It is now generally agreed that whenever possible, pancreatic secretion should be returned to the intestine.[12, 15] The question then arises as to what type of pancreatico-enterostomy is preferable. Among those described in patients there are few follow-up autopsy examinations, but several experimental studies on this problem have been reported.

Coffey (1909) attempted in dogs to implant the cut end of the pancreas into the jejunum, either end-to-end or end-to-side. He found the lumen of the bowel too small for this procedure, and he therefore devised a common lumen in a loop of jejunum, permitting good inversion of the bowel wall around the gland. Sweet and Simons (1915) implanted the cut end of the pancreas into the bowel successfully in dogs, without using Coffey's double lumen. Examination after four to six weeks showed the lumen to be patent in five dogs, whereas in three others the normal ducts reopened around the ligatures and the implanted end closed over. Patrie and associates (1917) stripped parenchyma from the ducts for a distance of three-fourths of an inch and pushed the duct into the bowel through a small hole. Implants were made into the colon as well as the small bowel. Of 16 dogs, three died of acute pancreatitis and two ducts closed over; the remaining ducts were open after four to 12 weeks.

Tripodi and Sherwin (1934) implanted the cut end of the pancreas into the stomach in dogs. Some of the ducts remained open after nine to ten months, and trypsin was found in the stomach. Person and Glenn (1939) performed similar experiments. They found that the intragastric part of the pancreas was digested after 20 to 25 days, but the ducts remained open, as shown by the presence of pancreatic lipase in the gastric juice, and by the injection of fluid through the anastomosis.

Poth (1944) devised a silver cannula, which he used successfully to implant the pancreatic duct in dogs and in a patient. The duct was attached to

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a flange, which was held in the bowel by means of a purse-string. Zinninger (1942) used a vitallium tube in a similar fashion in one patient, who did well. Varco (1945) used a small rubber catheter stretched on a stylet and allowed to expand inside the duct, to obtain a tight fit. The method has been used successfully in dogs and in several patients. It is described more fully below.

Smithy and associates (1945) implanted the cut end of the uncinate lobe of the pancreas into the seromuscular layer of the bowel in dogs, without opening the submucosa. They used a necrotizing suture, passing through the ligated duct and the sub-mucosa of the bowel, as described by Cattell,[2] in seven of the 22 animals studied. Patency was determined after seven to 48 days by observing the opened duodenum while secretin was injected. Fifteen ducts were open, including six of the seven in which a necrotizing suture had been used. A large retention cyst developed in one dog.

Wells and Annis (1949) examined dogs 15 to 66 days after implanting the cut end of the pancreas into the stomach. Amylase was found in the stomach in four of the six dogs surviving operation. Three had some pancreatic atrophy. Microscopic section of the specimen obtained at 66 days showed healing of the gastric and duct mucosa. These authors suggest that it is safer to implant the pancreas into the stomach than into the intestine, because activation of the pancreatic enzymes at the site of healing is thus avoided.

It is difficult to judge, from the foregoing review, whether any of the procedures described is preferable to the others. In the experiments to be related, an attempt was made to compare the final results of several types of pancreatico-intestinal anastomosis performed in dogs. In this animal the pancreas is mainly enclosed in the duodenal mesentery, whereas the human gland lacks serosal covering except in a few small areas. In the dog there are two main pancreatic ducts, a small one adjacent to the common bile duct, and a larger one entering the duodenum 3 or 4 cm. lower down. Probably a number of very tiny ducts enter the duodenum from individual lobules. The two main ducts nearly always communicate within the gland.[7] The larger of the two will usually accommodate a tube 2.5 mm. in outside diameter, and is 5 to 8 mm. in length from its main bifurcation to the duodenum. The human duct is about 3 mm. in diameter in the head and body of the gland.[1] It may be much larger when obstructed. In both species the ducts are surrounded by a layer of dense connective tissue which holds sutures well.

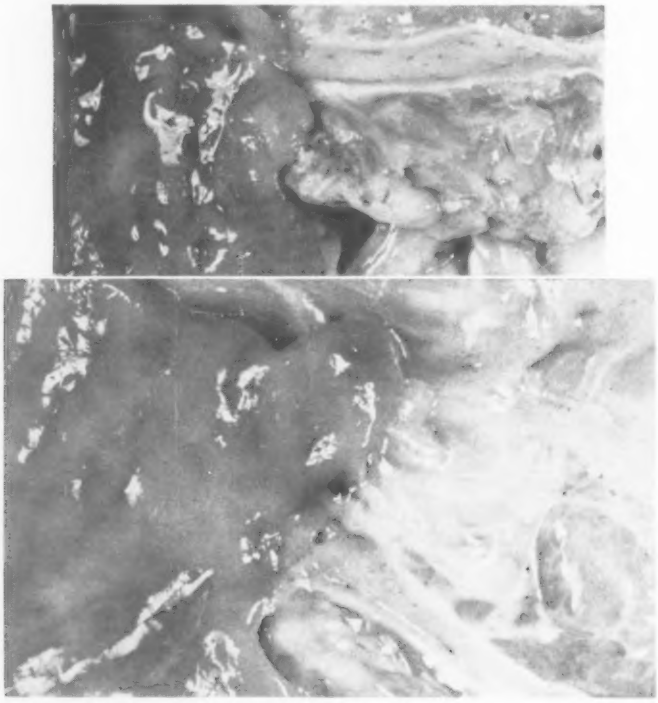
METHODS

Operations were performed on 65 dogs of average or large size. The smaller pancreatic duct, entering the duodenum at the papilla of the common bile duct, was ligated and divided in every case, because previous experiments had indicated that an anastomosis was more likely to close if normal channels remained open. In all but the experiments described in Group VIII, the main duct was divided at the level of the submucosa, and the duodenal side was closed. The duct was re-anastomosed to the duodenum at a convenient site,

about 10 cm. below its original location, by the various means to be described. Penicillin, 25,000 units, was instilled into the operative area before closure.

The animals received a standard diet beginning on the day after surgery. Early feeding doubtless provoked a flow of pancreatic juice and put the anastomoses to a test of leakage. Most of the dogs survived in good health, and were used for other unrelated experiments, until sacrificed. The foamy stools typical of pancreatic deficiency did not occur even when the anastomosis proved to be completely obstructed.

A



B

FIG. 1.—(A) Photograph of opened anastomosis, dog 14, 112 days after operation; anastomosis with interrupted 6-0 silk, with a tube. The pancreatic duct enters from the right, and a buttress suture is visible in the lumen of the duodenum. x 1.5. (B) Photograph of opened anastomosis from dog 1, 233 days after operation. Anastomosis with interrupted 6-0 silk. The duct enters the picture from the lower right, and the opened duodenum is on the left. A buttress suture of 4-0 silk, and one of the anastomotic sutures are visible. x 1.5.

At autopsy the main pancreatic duct in the body of the gland was cannulated, and connection was made to an L-tube, the vertical limb of which was filled with water to a height of 20 cm. Water was allowed to run into the intestine until it stopped, when the remaining level was noted. This is recorded in the tables as a measure of patency of the anastomosis. Occasion-

ally some sticky mucus had to be washed out of the duct before water would flow through it, a circumstance also noted in ducts that had not been operated on. The ducts were then opened and the circumference was measured at the widest part. Microscopic sections were made through the anastomosis, and of the parenchyma.

EXPERIMENTS AND RESULTS

Three general types of pancreatico-enterostomy may be distinguished: (1) direct anastomosis by suturing the end of the duct to the mucosa of the bowel; (2) various methods of implanting the duct through the intestinal wall; and

TABLE I.—*Summary of 16 Suture Anastomoses of the Pancreatic Duct to the Duodenal Mucosa. In the Second Group of Eight Dogs, a Tube Was Placed Through the Anastomosis.*

						Findings in Pancreas			
								Parenchyma	
		Dog No.	Days Lived Postop.	Cause of Death	Patency cm. H_2O	Size of Anas.	Circumf. of Duct	Normal*	Necrosis	Fibrosis
Group	I†	1	233	Sacr.	0	Wide	4.5 mm	X		
		2	3	Pancreatit.	..	Open, kink	...		X	
		3	42	Pneum.	0	Wide	4.5	X		
		4	85	Sacr.	0	Wide	4.0	X		
		5	167	Sacr.	0	Wide	3.5	X		
		6	16	Distemp.	0	Wide	4.0	X		
		7	70	Sacr.	14	Small	7.0			X
		8	102	Sacr.	0	Wide	5.0	X		
Group	II‡	9	264	Sacr.	0	Wide	5.0			X (Ligation 2 wks. prior to anas.)
		10	99	Sacr.	0	Wide	3.5	X		
		11	68	Sacr.	0	Wide	3.5	X		
		12	54	Sacr.	0	Wide	4.0	X		
		13	91	Sacr.	0	Wide	5.0	X		
		14	112	Sacr.	0	Wide	4.0	X		
		15	87	Sacr.	0	Wide	5.0	X		
		16	71	Sacr.	0	Wide	4.0	X		

* "Normal" indicates parenchyma of that dog's pancreas was normal grossly and microscopically, etc.
† Anastomosis.
‡ Anastomosis with tube.

(3) implantation of the entire cut end of the pancreas. Within these types we have investigated eight of the possible methods, using eight dogs in each group except the last.

Group I.—Direct suture anastomosis. The unopened duodenum was united to one side of the pancreas adjacent to the divided duct by several interrupted sutures of 4-0 silk. A very small opening was then made into the bowel and the protruding mucosa was clipped off. The end of the duct was sutured to the mucosa (including submucosa) around the opening, using from seven to nine interrupted sutures of 6-0 silk in a single row. The duodenum and pancreas were then united all around the anastomosis with more sutures of 4-0 silk.

Results are indicated in Table I. One dog died of pancreatitis after three days, apparently due to kinking of the duct near the anastomosis by one of the

buttressing sutures. There appeared to be no obstruction of the anastomosis itself, as water passed through it readily. One other dog had some pancreatic atrophy and fibrosis, with a narrowed anastomosis. The remaining dogs died or were sacrificed at intervals of 16 to 233 days. The anastomoses were patent and the glands were normal grossly and microscopically (Fig. 1B).

Group II.—Direct suture anastomosis with tube. This operation was the same as the preceding. In addition, when the anastomosis was half completed, a piece of polythene tubing 2 cm. long and 2.5 mm. in external diameter was inserted into the duct and tied in place by a suture passing through the end of the duct. The other end of the tube was then inserted into the opening in the duodenum.

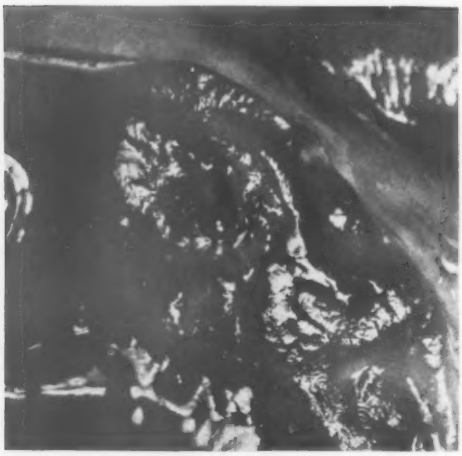


FIG. 2.—Photograph of specimen from dog 9, 264 days after operation; anastomosis with interrupted 6-0 silk, and a tube. The duct orifice is seen at its juncture with the duodenal mucosa. x 1.4.

One tube was found in the cage on the third day, and another on the twelfth; the others were not observed, but none were present at autopsy. The dogs were sacrificed at intervals of 54 to 264 days after operation. The results were good in all the dogs (Table I; Figs. 1A and 2).

Group III.—Simple implantation of the duct. A suture was passed through one side of the end of the duct and used to pull it into the duodenum; the suture passed through the opposite wall of the intestine and was fastened there. Interrupted silk was used to attach the pancreas to the duodenum all around the site of implant.

Results are indicated in Table II. Several ducts were found protruding 2 or 3 mm. into the bowel. The holding sutures had all disappeared. One dog died of leakage, and one had only a pinhole opening in the duct; the others, observed 22 to 185 days after operation, showed satisfactory anastomoses.

Group IV.—Implantation with a tube. Polythene tubes 2 cm. long and from 2.0 to 2.5 mm. in external diameter were fastened into the duct by a suture passing through the end of the duct and tied around the tube just distal to the end of the duct. Tube and duct were pushed through a small hole in the duodenum, and the pancreas was fastened in place in the usual manner.

Findings are listed in Table II. There were no deaths, but only four had an entirely satisfactory result, from nine to 174 days after operation. In one other dog the tube was still present at 15 days.

Group V.—Implantation over a very small tube. The reason for this experiment was the possibility that a snugly fitting tube might induce fibroplasia

and stenosis of the duct. It was thought that a small, loosely fitting tube would serve as a splint and as a channel for the fluid, with less disturbance of the healing process. The procedure was the same as the preceding, except that the tubes were 1 mm. in outside diameter.

As may be seen in Table II, there were only four good results, and one death occurred from leakage.

TABLE II.—*Summary of 24 Experiments in Which the Pancreatic Duct Was Implanted into the Duodenum.*

				Findings in Pancreas					
	Dog. No.	Days Lived Postop.	Cause of Death	Patency cm. H_2O	Size of Anas.	Circumf. of Duct	Parenchyma		
							Normal	Necrosis	Fibrosis
Group III Simple Implant	17	44	Pneum.	10	Pinhole	...	X		
	18	36	Sacr.	0	Wide	4 mm	X		
	19	185	Sacr.	0	Wide	4.0	X		
	20	22	Distemp.	0	Wide	4.0	X		
	21	24	Heat str.	0	Wide	5.5	X		
	22	3	Peritonit.	..	Leak	...		X	
	23	83	Sacr	0	Wide	4.5	X		
	24	170	Sacr.	0	Wide	4.0			X
Group IV Implant, tube	25	74	Sacr.	0	Wide	4.5	X		
	26	174	Sacr.	4	Wide	3.5	X		
	27	15	Pneum	..	Tube	...	X		
	28	60	Sacr.	2	Small	4.5	X		
	29	21	Sacr.	4	Small	4.0	X		
	30	47	Distemp.	0	Wide	4.0			X (Sl.)
	31	9	Eviscerat.	0	Wide	4.0	X		
	32	48	Cause undet.	10	Small	7.0			X
Group V Implant, Small Tube	33	189	Sacr.	..	Closed	...	X (Accessory duct)		
	34	246	Sacr.	4	Wide	4 0	X		
	35	152	Distemp.	0	Wide	4.5	X		
	36	163	Sacr.	0	Wide	4.5	X		
	37	8	Heat str.	..	Tube	...	X		
	38	96	Sacr.	8	Small	8.0			X
	39	131	Sacr.	0	Wide	4.5	X		
	40	5	Peritonit.	..	Leak	...		X	

Group VI.—Aseptic implantation by means of a necrotizing suture. This method has been described and used by Cattell.[2] In the present experiments the end of the duct was tied with 4-0 silk. A suture of the same material was passed through the duct proximal to the tie, and through the submucosa of the duodenum in the center of an area where the serosa and muscularis had been incised and spread open. The suture was tied as tightly as possible, and the pancreas was sutured to the cut edges of the seromuscular layer.

There was no immediate mortality in the eight dogs, but only two of the anastomoses were entirely satisfactory (Table III). One dog died, after 85 days, of bowel obstruction due to a cyst 7 cm. in diameter at the site of implant (Fig. 3). The duct was still in place, but did not open into the bowel. The pancreas around the duct was eroded, and pancreatic secretion probably filled the cyst from this area.

Group VII.—Implantation by Varco method. This procedure is designed to prevent any leakage of pancreatic juice at the anastomosis and to carry the juice well away from the area of healing. A rubber catheter several centimeters long and of suitable diameter is stretched over a stylet which protrudes into the wall of the catheter at one end. This reduces the external diameter of the tube so that it can be inserted into a duct of somewhat smaller diameter. The stylet is then removed and the tube expands into a tight fit; additional

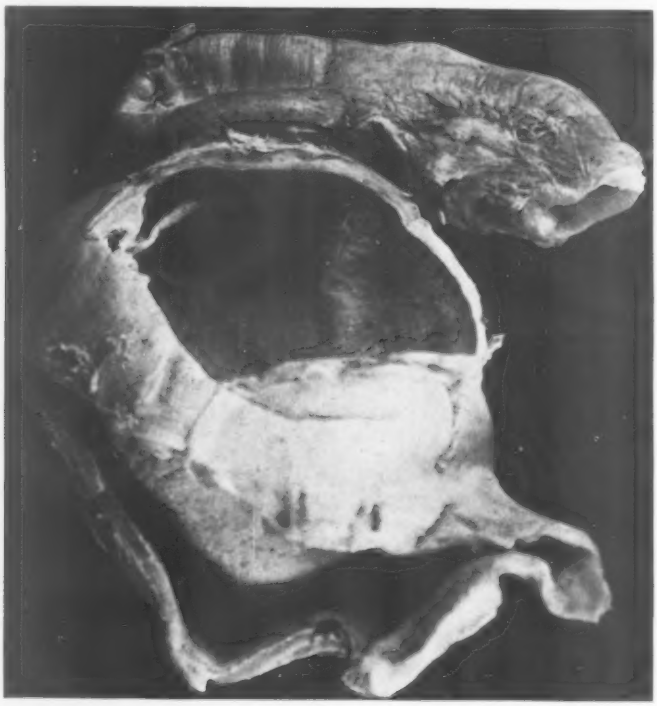


FIG. 3.—Photograph of specimen from dog 43, 85 days after implant of duct by necrotizing suture. The duodenum curves around the cyst in the shape of a C, and has been opened below and near the site of implant. The fibrotic remnant of the duct is visible in the top of the cyst, where there is an ulcerated area in the pancreas. x 0.8.

fixation is provided by a tie around the end of the duct. Implantation is then carried out as described for Group IV.

There were two deaths caused by leakage (Table III). The ends of the ducts in these dogs were necrotic when examined on the fifth postoperative day, and this may have been due to the use of too large a catheter. Good results were obtained in four dogs, examined 40 to 152 days after operation.

Group VIII.—Implantation of the cut end of the pancreas. In two dogs the upper pancreatic duct was ligated and divided, the gland was divided just above the lower duct, and the lower cut end was closed, leaving the lower part of the pancreas to drain through the large duct. The cut end of the upper part

of the pancreas was then implanted into the posterior wall of the antrum of the stomach, using two rows of interrupted 4-0 silk to invert the gastric wall around the pancreas. Examination at 42 and 48 days showed that the intragastric pancreas had been largely digested away and the area was covered by a thin layer of gastric mucosa over most of its extent. The openings of the

TABLE III.—*Summary of 21 Experiments in Which the Pancreatic Duct or the Cut End of the Pancreas Were Implanted by Special Methods.*

				Findings in Pancreas					
	Dog No.	Days Lived Postop.	Cause of Death	Patency cm. H_2O	Size of Anas.	Circumt. of Duct	Parenchyma Normal	Necrosis	Fibrosis
Group VI	41	170	Sacr.	2	Small	4.0 mm	X		
Necrotizing	42	185	Sacr.	19	Small	4.0	X		
Suture	43	85	Cyst, obstr.	..	Closed	...			X (Large retention cyst)
	44	35	Cause undet.	..	Closed	9.5			X
	45	40	Sacr.	..	Closed	7.0	X		
	46	109	Sacr.	4	Small	5.0	X		
	47	56	Sacr.	.0	Wide	4.5	X		
	48	85	Sacr.	0	Wide	4.0	X		
Group VII	49	109	Sacr.	9	Small	4.0	X		
Varco	50	5	Peritonit.	..	Leak	...		X	
	51	60	Sacr.	0	Wide	4.0	X		
	52	5	Peritonit.	..	Leak	...	X		
	53	62	Sacr.	20+	Pinhole	10.0			X
	54	33	Sacr.	6	Wide	6.0	X		
	55	152	Sacr.	0	Wide	4.0	X		
	56	40	Sacr.	0	Wide	4.0	X		
Group VIII									
Implant,	57	9	Eviscerat.	..	Closed	...		X	
Jejunum	58	94	Sacr.	0	Wide	4.0	X		
Implant,	59	42	Sacr.	14	Pinhole	7.0	X		
Stomach	60	48	Distemp.	20+	Pinhole	6.0			X
Implant, Duodenum	61	14	Duod. obstr.	..	Closed	...			X

ducts were of pinhole size, with dilatation behind, and induration of one of the glands (Table III).

In one dog the cut end of the lower part of the pancreas was implanted into the side of the duodenum. This animal died of bowel obstruction at the site of implant, after 14 days. A large mass of scar and indurated pancreas closed the duct.

In two dogs the cut end of the lower pancreas was implanted into the distal end of the divided jejunum; bowel continuity was established by an end-to-side anastomosis about 20 cm. lower. One dog died on the ninth day. There was a mass of scar and induration which closed the end of the duct. The other dog showed a good result when sacrificed after 94 days (Table III).

COMMENT

The amount of fibrosis and atrophy of the pancreas, apparent grossly as induration and shrinkage, was usually proportional in these experiments to the amount of ductal obstruction present. There was little to suggest that a

freely patent duct permitted injury of the gland through a reflux of intestinal contents.

From the point of view of safety and the best end result, it appears that a suture anastomosis is preferable, at least to the other methods we have tested.

The amount of time required to make a suture anastomosis is only a few minutes more than the time necessary to perform any type of implant, or even to close the end of the pancreas. Since the duct in the dog is smaller than the normal human duct in the head and body of the gland, anastomosis by suture should be feasible in most patients who are not obstructed as well as in those who are. The end result to be desired is a rapid healing of the duct epithelium to the mucosa of the gut, and there is probably no shortcut better than suturing the two structures carefully in apposition.

SUMMARY AND CONCLUSION

Eight methods of pancreatico-intestinal anastomosis have been tested in 61 dogs. Direct suture of the pancreatic duct to an opening in the mucosa of the intestine, with the use of a temporary tube through the anastomosis, gave the best results in these experiments.

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NONOPERATIVE TREATMENT OF PERFORATED DUODENAL ULCER

PRELIMINARY REPORT OF 16 CONSECUTIVE CASES WITH NO MORTALITY*

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PERFORATION IS THE MOST DEADLY COMPLICATION of peptic ulcer. Until recently, surgical closure of the perforation has remained the unchallenged treatment of choice. This is a preliminary report of 16 consecutive cases treated without operation. In this series the mortality rate is zero and only one complication was encountered.

This treatment was instituted in October, 1945 on the following rationale: That (1) the mortality rate of operative treatment was undesirably high; (2) the incidence of complications and prolonged morbidity with operative treatment was alarmingly high; and (3) peritonitis *per se* was no longer the killer of former years, because, with the aid of the newer armamentarium at our disposal, the peritoneum usually will localize and absorb the contaminant. Therefore, if these patients could be treated nonoperatively with equal or, as we hoped, a lowered mortality and a lowered incidence of complications, they were being subjected to an unnecessary operation in surgically closing the perforation.

REVIEW OF LITERATURE

The literature abounds with excellent discussions of diagnostic criteria of perforated peptic ulcer, as well as the bacteriology of the peritoneal cavity in such cases and late results following perforation. They need no discussion here.

The mortality rates of series treated surgically after 1940 and a series of 15,340 cases collected by DeBakey[6] in his review of the literature on this subject from 1930 to 1940 is given in Table I. The mortality rate since the advent of the antibiotics and improved postoperative treatment has shown a significant drop, but in most reported series it is still undesirably high.

The incidence of complications following operative treatment is very high, thus resulting in prolonged hospital stay (see Table II).

Perhaps the earliest report of recovery of a perforated peptic ulcer without surgical treatment was recorded in 1870 by Redwood.[12] In 1871 Ross[13] and

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* This series was started at the suggestion and under the supervision of Dr. Edmund Horgan, Winchester, Virginia, formerly Colonel, MC, AUS, while he was chief of the Surgical Service of Brooke General Hospital, Fort Sam Houston, Texas. A portion of the series was treated under the supervision of Col. Sam F. Seeley, MC, USA. Submitted for publication October, 1949.

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Tinley[17] each added a case of perforation with recovery. Hall[8] added another case in 1892.

Wangensteen,[19] in 1935, reported four patients treated nonoperatively, with one death. He recently stated that he had given up treatment by other than surgical means.

Bedford-Turner,[3] in 1945, reported a series of six patients with perforations treated without surgery. There were no deaths and no complications. All

TABLE I.—*Mortality of Perforated Peptic Ulcer: Surgical Treatment.*

	Year	Total Cases	Mortality in Percent
DeBakey (collected cases)	1930–1940	15,340	23.4
McClure	1940	91	7.7
Ross	1940	175	16.0
DeBakey & Odom	1940	209	18.2
Davidson & Rudder	1940	155	28.0
Cohn	1941	300	15.0
Berson	1942	151	15.2
Donald & Barkett	1942	116	18.5
O'Donoghue & Jacobs	1942	200	24.5
Barber & Madden	1943	86	12.8
Paletta & Hill	1943	85	16.9
Timoney	1943	246	19.5
McCabe & Mersheimer	1943	87	25.2
Estes & Bennett	1944	80	8.7
Raw	1944	312	14.4
Black & Blackford	1945	93	12.0
Baritell	1946	88	1.1
Graham, R. R.	1946	130	6.4
Shipley & Walker	1935–1946	188	20.0
Luer	1938–1948	318	18.2

patients had duodenal ulceration demonstrated by roentgenography after recovery. The patients were all males, three between the ages of 20 and 30 years, and three between 60 and 65 years.

Taylor[16] recently reported a series of 28 consecutive cases of perforated peptic ulcers. Of these, 24 were treated nonoperatively with intermittent gastric suction with three deaths. One death was due to pulmonary embolism, one

TABLE II.—*Complications with Surgical Treatment.*

	McCabe and Mersheimer		Shipley and Walker		Odom and DeBakey		Luer		DeBakey Collected Cases	
Total Cases in Series	88		188		211		318		772	
	Cases	%	Cases	%	Cases	%	Cases	%	Cases	%
Pulmonary affections	24	27.0	41	21.8	24	11.3	54	17.0	..	32.8
Wound infections	27		24	12.7			75	26.7		
and		42.0			61	28.9			..	25.4
Eviscerations	10		6	3.2			9	2.8		
Generalized peritonitis	8	9.0	17	9.0	26	19.8	29	9.1	..	24.7
Abscess—intraperitoneal, subphrenic, etc.	2	2.2	7	3.7	16	7.6	31	9.7	..	7.2
Fistulas	2	2.2	1	0.5	..	....	..	....	..	1.6
Ileus	..	....	3	1.6	5	2.3	..	....	..	1.6
Hemorrhage	..	....	3	1.6	3	1 4	3	0.9	..	1.4
Embolus	..	....	1	0.5	..	....	..	....	..	1.2
Miscellaneous	..	....	..	....	..	....	7	2.2	..	4.1

to the moribund state of the patient on admission and one to generalized peritonitis.

Visick[18] reported a series of 14 consecutive, unselected cases of perforated peptic ulcers treated by gastric suction. All of his patients were men. Twelve had duodenal ulcers, one a gastric ulcer and one a stomal or gastrojejunal ulcer. There were three deaths in this series, one following surgical intervention. One patient developed a subphrenic abscess which required surgical drainage on the tenth day.

Baritell[2] reported an 88 case series treated surgically and an additional five selected cases which he treated nonoperatively because their histories indicated that the perforations were more than 12 hours old, and the patients seemed to be improving clinically. All five recovered. One patient developed a subphrenic abscess which was drained surgically.

Birks[5] reported a series of nine selected cases treated nonoperatively with no deaths or complications.

Bingham[4] reported five presumably selected patients with no deaths or complications.

It is impossible to compare accurately or consolidate all reports of nonoperative treatment because of differences in the treatment routine. Some series are of consecutive cases, some of selected cases. A few surgeons used intermittent gastric suction; more used continuous. The exact routine, with special reference to antibiotic and/or chemotherapy, is not stated in some reported series. Two authors include in their series patients who were operated upon, with death directly attributable to surgical intervention. Table III consolidates these reports of nonoperative treatment.

TABLE III.—*Consolidation of Nonoperative Treatment of Perforations.*

Author	Cases	Consecutive or Selected	Deaths	Complications
Bedford-Turner	6	Unstated	0	0
Taylor	24	Consecutive	3	1 generalized peritonitis—died 1 pulmonary embolus—died
Visick	13	Consecutive	2	1 subphrenic abscess, recovered
Baritell	5	Selected	0	1 subphrenic abscess, recovered
Birks	9	Selected	0	0
Bingham	5	Selected	0	0
This series	16	Consecutive	0	1 subphrenic abscess, recovered
Totals	78		5 (6.4%)	5 (6.4%)

OUTLINE OF TREATMENT

When a suspected case of perforated peptic ulcer is admitted to the hospital and the diagnosis established, constant gastric suction is immediately instituted via Levin tube. Constant gastric suction is the most important single element in the treatment. The importance of constant watching of the suction apparatus and Levin tube to keep the system functioning cannot be emphasized too greatly. These patients require the care of an experienced nurse who will check

the apparatus at least every half hour. These cases should be supervised by a surgeon experienced in the care of patients with peritonitis and who will frequently search for complications and promptly and adequately treat them if they arise. The surgeon should examine these patients at least every four hours during the first two days of this treatment. Nonoperative treatment of perforated peptic ulcer cannot be handled casually.

Laboratory determinations required are: Blood chloride level, carbon dioxide combining power, plasma protein and hematocrit, complete blood counts and serum amylase determination. An intake and output record is maintained. These procedures provide "base lines" for future determinations and act as controls to aid us in our decision as to types and amounts of intravenous fluids to be given.

Sodium sulfadiazine, 2 Gm., is given intravenously immediately and 1 Gm. every four hours thereafter. Penicillin, 50,000 to 100,000 units, is given intramuscularly every three hours. Absolutely nothing is given by mouth.

During the first day the patient receives 3000 cc. of fluid intravenously on an empiric basis, provided there are no contraindications such as cardiac or renal disease. These fluids consist of 1000 cc. of 5 per cent glucose in distilled water, 1000 cc. of one-sixth molar sodium lactate solution and 1000 cc. of casein hydrolysate; 1000 cc. of physiologic saline solution are substituted for one of the above as indicated by blood chloride levels. Each succeeding day protein as casein hydrolysate, whole blood or serum albumen, in significant amounts to spare the protein stores is given. The amount and types of the fluids administered on subsequent days are determined by the blood chemistry determinations and intake and output record.

Thiamin chloride, 100 mg.; nicotinamide, 100 mg.; and ascorbic acid, 300 mg., are given daily in intravenous solutions.

Milk and cream mixture is started by mouth on the fourth to sixth day. This is given one ounce every hour with the Levin tube in place but clamped off. The patient is carefully watched for signs of peritoneal irritation. If he tolerates this feeding well for a day, and all of our patients have, the tube is withdrawn. The diet is gradually increased and after ten days to two weeks the patient is transferred to the Medical Service for treatment as any uncomplicated acute peptic ulcer patient.

It is extremely gratifying to observe patients under this regimen. Very soon they are comfortable and usually require no more than one or two doses of morphine. Rigidity regresses rapidly, disappearing from below upward, and is usually gone within 24 to 48 hours. Residual tenderness may persist for four to six days. Peristalsis returns in from one to three days.

The fact that these patients do not seem so ill as those previously treated by surgical closure, and their rapid and smooth recovery, is most impressive.

DISCUSSION OF RESULTS

Since September, 1945, 22 patients were seen who would have been subjected to surgical intervention as perforated peptic ulcers prior to the institution

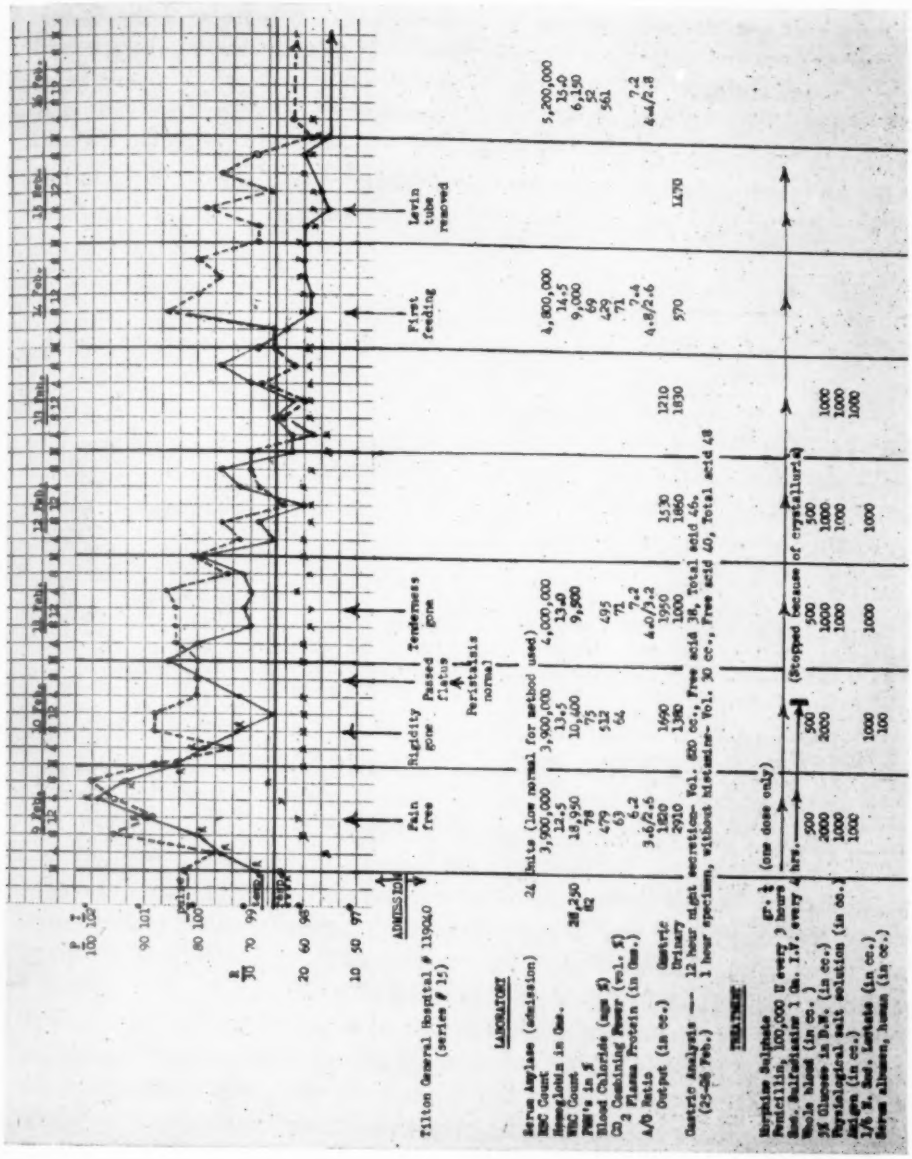

FIG. 1.—Chart showing typical course, laboratory findings and treatment (Case 15 of series).

of this regimen, it is believed. All 22 of these patients were treated nonoperatively in the manner described above with no deaths. The one complication encountered, a subphrenic abscess, was drained surgically on the tenth day after perforation. This patient then made an uneventful recovery.

Six of these 22 patients were eliminated from this series because free intraperitoneal air was not demonstrated. They were assumed to be of the "penetrating" or *formes frustes* type of perforation. These six patients all recovered without complication.

The remaining 16 patients all had history and physical findings definitely pointing to perforation of a peptic ulcer. All had duodenal ulceration demonstrated by roentgenography after recovery. Thirteen of these 16 patients had free intraperitoneal air demonstrated by roentgenograms. The other three

TABLE IV.—*Summary of Present Series.*

Case No.	Date of Admission	Age	Duration of Ulcer Symptoms	Food Perforation Interval	Perforation Treatment Interval	Free Intraperitoneal Air	Complications
1. 366706	16 Oct. 45	38	2 years	4 hours	5 hours	Yes	None
2. 382006	15 Mar. 46	23	6–8 mos.	18–20 hours	3 hours	Yes	Supbhrenic abscess
3. VA 2386	21 Mar. 46	40	2 years	½ hour	11 hours	Yes	None
4. 386104½	12 May 46	40	14–15 yrs.	3 hours	3 hours	No air, previous perforation	None
5. VA 3100	21 June 46	33	.....	.....	.....	Yes	None
6. VA 3962	5 Sept. 46	21	1 year	1 and 4 hours	1 hour	Yes	None
7. VA 4385	15 Oct. 46	48	3 years	12 hours	2½ hours	No air, previous perforation	None
8. 397852	8 Nov. 46	26	3 years	9½ hours	½ hour	Yes	None
9. VA 5408	21 Jan. 47	24	3–4 years	2 hours	2½ hours	Yes	None
10. VA 5788	18 Feb. 47	23	1½–2 years	4 hours	66 hours	Yes	None
11. VA 5957	5 Mar. 47	25	1 year	9 hours	3½ hours	No air, previous perforation	None
12. VA 7695	7 July 47	36	2 years	1½ hours	4 hours	Yes	None
13. VA 8032	28 July 47	52	25 years	2 hours	64 hours	Yes	None
14. 70903	16 Dec. 48	29	5 years	3½ hours	5½ hours	Yes	None
15. 119040	8 Feb. 49	19	8 mos.	4 hours	3 hours	Yes	None
16. 121832	29 Apr. 49	45	?	8 hours	30 hours	Yes	None

patients had no free air demonstrated but did have a history of a previous perforation treated by surgery. Two of these three patients stated as their presenting complaints that their ulcer had ruptured again. We believe the diagnosis to be unquestionably established in these 16 patients.

Figure 1 is a graph of the clinical course of a typical patient for the first week of treatment. Sulfadiazine is usually given until the patient is afebrile, but, because this patient had crystalluria, the drug was discontinued to avoid complications. This patient, because of anemia, received much more whole blood than usually is used. As a rule, plasma protein levels can be kept well up in normal range by casein hydrolysate. This graph includes all major treatments, laboratory findings and clinical course to illustrate the regimen. Roentgenograms taken three weeks after perforation demonstrated a duodenal ulcer with markedly deformed duodenal bulb.

Figure 2 is a photograph of this man's admission roentgenograms in upright and left lateral decubitus positions, and shows free intraperitoneal air.

Table IV represents this series. Attention is called to the ages of the patients, varying from 19 to 52 years. The interval between last food ingestion and perforation varied from 30 minutes to 20 hours. The time interval between perforation and institution of treatment varied from 30 minutes to 66 hours.

Figure 3 is interesting because it represents the largest collection of free intraperitoneal air seen in this series. The roentgenograms shown were taken on admission, three, seven and nine days after perforation, and show the rapid absorption of air. On the tenth day no air was seen.

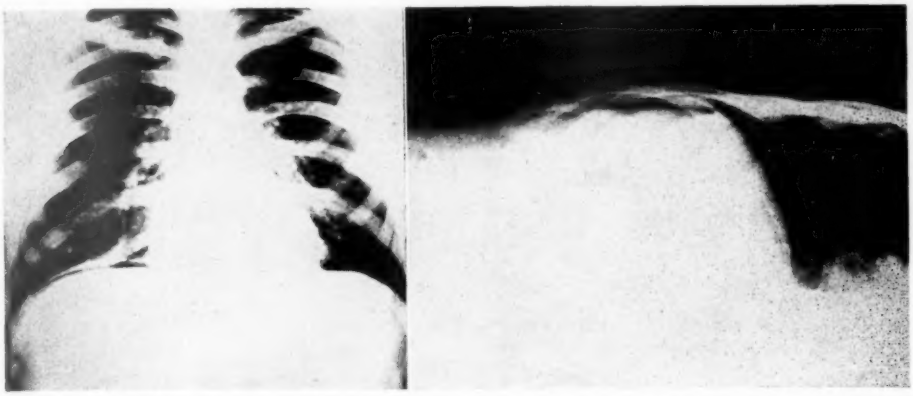

FIG. 2.—Admission roentgenograms of Case 15 in upright and left lateral decubitus positions showing free intraperitoneal air.

The author has not used the word "conservative" in reference to the nonoperative treatment presented here because it is believed that such treatment should still be considered radical today. Radical treatment seems justified, however, if in that way the patient can *safely* be spared an operative procedure.

By eliminating surgery most of the complications can be avoided. With no operative wound there can be no wound infection, evisceration or fistula. By eliminating anesthesia the pulmonary complications should be minimized. It is necessary to be concerned then with peritonitis, intraperitoneal abscess formation, ileus, hemorrhage, pulmonary embolus and the incidental pulmonary conditions. The complications reported in all series treated nonoperatively total five (see Table III). Three patients had subphrenic abscesses which responded well to surgical drainage. Two patients died of complications, one of generalized peritonitis and one of pulmonary embolism. Apparently subphrenic abscess is the most common complication encountered and must be watched for carefully. It is not felt that this complication is more frequent with nonoperative treatment than with surgical treatment.

That rest is the body's method of treating peritonitis is generally conceded. The old Ochsner method of treating peritonitis following appendiceal rupture

is an application of this principle. Certainly nonoperative treatment of perforated amebic ulcers of the colon results in much lower mortality. These two examples cited are not the same as perforated peptic ulcers but they do represent instances of gross contamination of the peritoneal cavity. They illustrate the prodigious tasks the peritoneum will perform in localizing and absorbing bacterial insults if left alone and allowed to work undisturbed. Localized collections of purulent material should, however, be drained surgically.

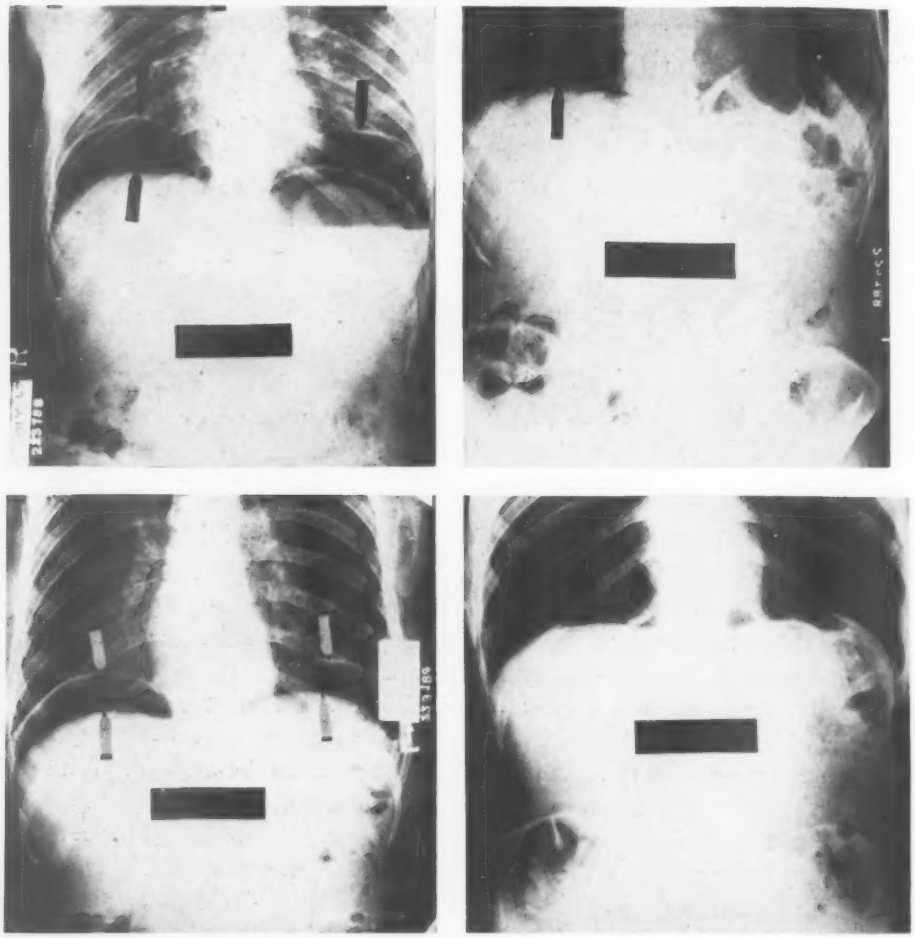

FIG. 3.—Roentgenograms of Case 12 showing the largest collection of free intraperitoneal air in this series. The upper left film was taken on admission; the lower left three days after admission; the upper right was taken seven days after admission and the lower right nine days after admission. On the tenth day no free air was demonstrable.

In the past ten years the medical profession has acquired potent chemotherapeutic agents and antibiotics which render many dread infections less fearsome. Our understanding of fluid and electrolyte balance of the body in pathologic conditions and our ability to correct imbalances has advanced

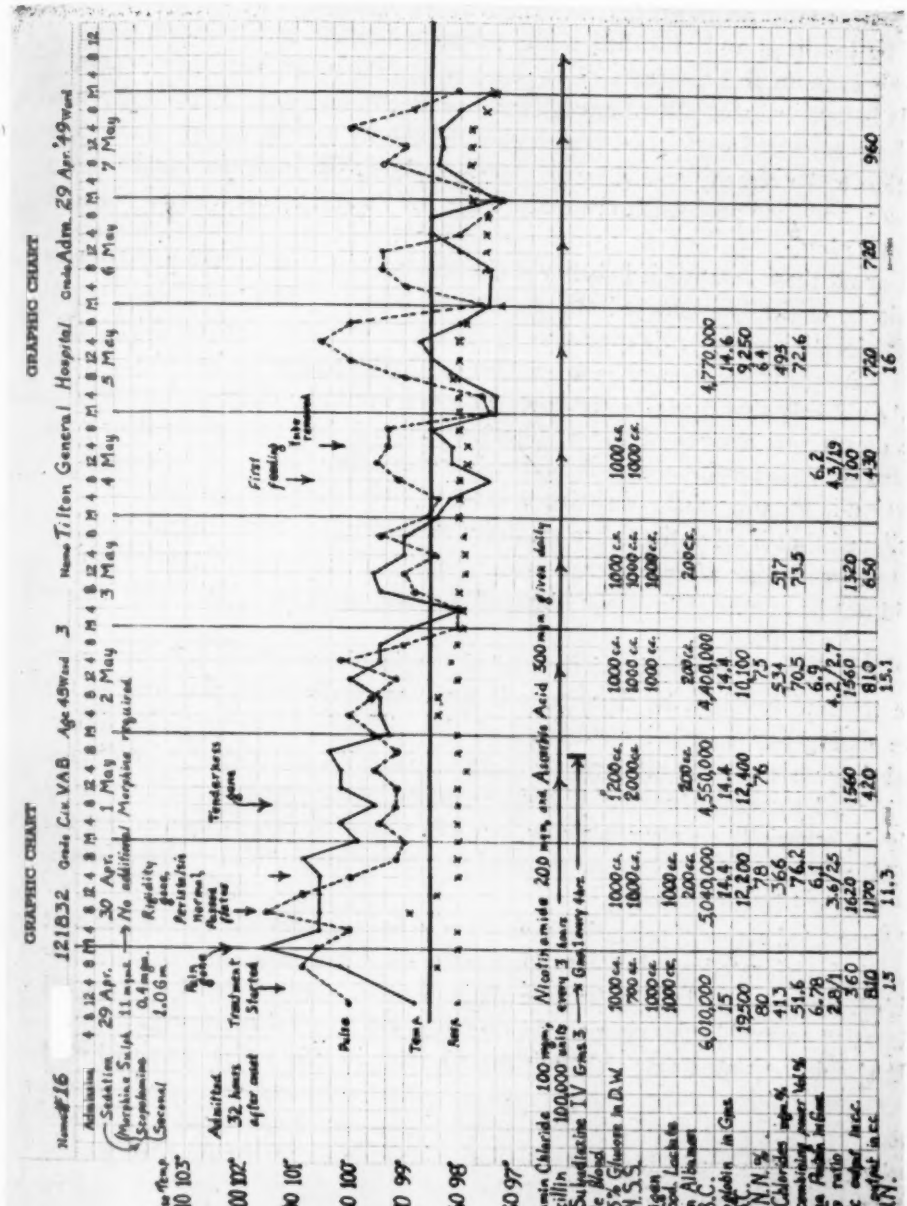

FIG. 4.—Chart showing clinical course of Case 16. This patient was admitted thirty hours after perforation and had pneumonitis on admission. This was subsequently proved to be tuberculous pneumonitis.

greatly. Almost all of the essential elements of a well balanced diet can be provided parenterally.

It seems logical to believe that chemical peritonitis and contamination by bacteria of mild pathogenicity, as occurs in perforation of peptic ulcers, could also be localized and absorbed if the peritoneal cavity is not invaded and if sealing is permitted to occur.

It is believed that all surgeons, after opening the peritoneal cavity for surgical treatment of perforated peptic ulcers, have frequently observed the perforation to be already sealed by an omental plug. The surgeon breaks down this seal and then proceeds to reproduce the body's seal by suture and often an omental plug.

For all these reasons it is believed the treatment of perforated peptic ulcer should be critically examined and re-evaluated.

Perforation of a gastric cancer must always be considered, even though this is an infrequent occurrence compared to perforation of a benign ulcer. It is highly unlikely that a perforated gastric cancer would be diagnosed grossly at time of surgical closure of the perforation unless it had metastasized or progressed to an incurable state. American surgeons do not consider that gastric resection is the procedure of choice to treat a perforation of a gastric ulcer. It is believed that if all patients are carefully studied with cancer in mind within two to three weeks after perforation the definitive treatment of this condition would not be appreciably delayed beyond the time consumed if the perforation is treated surgically.

An erroneous diagnosis of perforated peptic ulcer, followed by nonoperative treatment, might be disastrous if the correct diagnosis was one of the acute emergency surgical conditions of the abdominal cavity. The differential diagnosis of perforated peptic ulcer is too well known to require discussion here. By means of careful history, examination by physical and laboratory methods, especially roentgenography, one can almost always rule in or out the possibility of some other intra-abdominal pathologic condition requiring immediate surgical intervention.

In reported series of operative cases diagnosis is correct in 95 to 98 per cent of cases of perforated peptic ulcer. Unless the diagnosis is definite and unquestionable, however, it is felt that the patient should be subjected to surgical exploration to avoid missing a condition requiring surgical treatment.

It should be noted that although the 16 cases in this series are unselected and consecutive cases, the roentgenographic examinations of the stomach and duodenum two to three weeks after perforation demonstrated that in all cases the ulcer was duodenal. How perforation of gastric ulcers would react to our treatment is unknown. No reason is known to indicate the results would be different. Theoretically it should be easier to keep the stomach empty than the duodenum. A Levin tube is used and it is not passed into the duodenum or pylorus. About four to six inches of the tube are in the cardia of the stomach. No safe way to determine the site of perforation before recovery without opening the peritoneal cavity is known.

SUMMARY

A series of 16 consecutive, unselected cases of perforated duodenal ulcers treated with constant gastric suction and antibiotics, without operation, is presented. There was no death in this series. One complication, a subphrenic abscess necessitating surgical drainage, was encountered.

Previous series of perforations treated nonoperatively are reported.

The treatment used for this series is discussed. A graph showing the clinical course of a typical case is presented.

Conclusions based on a series of this size are unjustified. It is believed that the results obtained justify continuance of this method until the series is large enough to warrant conclusions.

It is necessary to emphasize again that this treatment should be under the supervision of a surgeon, that the treatment must not be considered lightly, that patients must be watched carefully and frequently, that diagnosis must be correct and that constant and adequate gastric suction is the most important factor in the treatment.

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BILIARY TRACT SURGERY IN A SMALL COMMUNITY HOSPITAL

A TEN-YEAR SURVEY*

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WHEREAS EXPERIENCES with biliary tract surgery in metropolitan and university hospitals have been adequately reported, there are few papers in the literature on this subject emanating from hospitals in small communities. Reports of surgery done in rural areas, although of limited inherent interest, seem justified on several grounds. Such reports offer data, which, when compared with those issuing from large centers, serve as a rough index of the degree to which surgeons in outlying parts have been able to avail themselves of the advances made by teaching institutions. With the current sociologic interest in medicine, which conjectures relatively unsatisfactory care for persons in less densely populated areas, accurate information would seem apposite. Although the urban surgeon has undeniable advantages over the rural one, the latter enjoys some favorable circumstances, as the results which follow attest—and particularly those results in acute cholecystitis. In a small community the total number of social unfortunates is small, and, instead of being cared for in charity outpatient departments, they are treated along with, and in the same manner as, private patients. Furthermore, in a small community there seems to be opportunity to see patients early in their disease.

MATERIAL

All operations performed upon the biliary tract in a hospital of 128 beds, serving a community of 20,000 persons and a surrounding countryside of approximately the same number of persons, during the decade ending May, 1948, were studied. The operations were done by four general surgeons, all of whom have had formal training in surgery and who comprise the surgical staff of the hospital.

In the early years, student nurses served as assistants at operation. Later, the surgeons assisted each other. During the last three years, the four surgeons have been formally associated in their practice, and an approved surgical residency has been established. The surgical staff has become associated with the University of Rochester School of Medicine, Rochester, N. Y., and the chief of staff holds an appointment as assistant professor of surgery in that

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institution, an interesting affiliation between a rural hospital and a teaching institution. These arrangements have resulted in routine consultation in difficult cases, excellent assistance at operation, and improved care for all patients before, during, and after operation.

GENERAL CONSIDERATIONS

A summary of the data appears in Table I. The 348 operations include all biliary tract surgery done in the Bradford Hospital, with the exception of four cholecystostomies, done in the early years by a surgeon who was nearing retirement. There were no deaths in the omitted cases, and most of them appear in this report as subsequent cholecystectomies. No patient was refused operation. One patient, who had a stricture of the distal end of the common bile duct which persisted after cholecystectomy and choledochostomy, tired of our efforts and was referred to Dr. Herman Pearse, Professor of Surgery at the University of Rochester School of Medicine, who transplanted her common duct into a loop of jejunum prepared in the Roux-Y manner, with a satisfactory result four years after operation. A second patient, who had had three chole-

TABLE I.—*General Considerations*

Total number of cases	348
Acute cholecystitis	85
Chronic cholecystitis	246
Carcinoma of gall bladder	4
Secondary choledochostomy	12
Traumatic rupture of gall bladder	1
Total number of deaths	7
Percentage mortality	2.0%

cystostomies for cholelithiasis, had a cholecystectomy performed by us in 1942. The gallbladder contained several stones; the caliber of the cystic duct was narrow; the head of the pancreas was indurated; the common bile duct was small and was not opened. The patient continued to have attacks of pain in her right upper abdomen. Dr. Herman Pearse operated upon her in the Bradford Hospital in 1943. A stricture was found at the distal end of the common bile duct and a vitallium tube was implanted into the duct with the distal end extending into the duodenum. This patient estimates her improvement at about 85 per cent, although she has had occasional attacks of pain in her upper middle abdomen.

The overall mortality of 2 per cent is not unduly high.

Stones were recorded as being found in 259 cases, or 75 per cent of the patients. Records in a small hospital, particularly prior to the coming of resident surgeons, leave a great deal to be desired, and it is the author's belief that stones were present in a higher number of cases than were recorded.

The ruptured gallbladder occurred in a young man who, while intoxicated by alcohol, was involved in an automobile accident and thrown against the dashboard. A cholecystostomy was performed, and he made a complete recovery.

ACUTE CHOLECYSTITIS

The data concerning acute cholecystitis is summarized in Table II. The 85 cases constitute 24 per cent of all biliary tract surgery, a slightly higher ratio than is usually reported. This discrepancy is probably explained by the the fact that a community hospital is apt to treat a high proportion of acute conditions in general.

With the exception of a few cases in the early years, our practice was to regard all cases of acute cholecystitis as emergencies; the patient's metabolic disturbances were corrected, in so far as possible, in 12 to 18 hours, and operation was performed. This policy, and the advantage of seeing the patient early in his disease, which the surgeon working in a small community seems to have, accounts, we feel, for the relatively satisfactory mortality (3.5 per cent), and for the fact that it was possible to perform 78 cholecystectomies (as

TABLE II.—*Acute Cholecystitis*

Number of cases	85
Percentage of total biliary operations	24%
Types of operations:	
Cholecystectomy	78
Cholecystostomy	7
Choledochostomies	24
Number of deaths	3
Percentage mortality	3.5%
Causes of death:	
Cardiac failure	2
Acute hemorrhagic nephritis	1

against seven cholecystostomies) and 24 choledochostomies for acute cholecystitis. In all cases where cholecystostomy was performed, a subsequent cholecystectomy was done.

When the patient is extremely ill, as in the occasional patient who is not seen early in his disease, or who is elderly, or has complicating diseases, we favor a cholecystostomy. One death, in a gravely ill woman, might have been avoided had we done a cholecystostomy instead of a cholecystectomy.

The three deaths were ascribed to cardiac failure in two cases, and acute hemorrhagic nephritis in the third. Autopsy was obtained only in the third case.

CHRONIC CHOLECYSTITIS

The data concerning chronic cholecystitis is summarized in Table III. Cholecystectomy was performed in all cases. One patient, who had had a partial cholecystectomy elsewhere for acute cholecystitis, had the remaining third of her gallbladder excised and stones removed from her common bile duct with a satisfactory result.

Four patients had cholecystoduodenal fistulas. One of these patients was admitted to the hospital for acute obstruction of her small bowel by a large gallstone, which she passed spontaneously while being prepared for operation.

In all cases the fistulas were repaired successfully. One patient in this group has had subsequent attacks of pancreatitis.

The mortality in 246 cases of chronic cholecystitis, treated by cholecystectomy with or without choledochostomy, was 1.2 per cent for the ten-year period and 0.7 per cent (in 139 cases) for the past five-year period.

TABLE III.—*Chronic Cholecystitis*

Number of cases	246
Number of deaths	3
Percentage mortality	1.2%
Deaths in past 5 years, 139 cases	1
Percentage mortality past 5 years	0.7%
Causes of death:	
Coronary occlusion	1
Cerebral hemorrhage	1
Pulmonary embolus	1

The three deaths were ascribed to coronary occlusion, pulmonary embolus, and cerebral hemorrhage. The patient who succumbed following a cerebral hemorrhage was particularly interesting. His temperature following operation until his death was very high, and his course resembled the so-called "liver death." Careful dissection at autopsy disclosed no injury to the hepatic

TABLE IV.—*Exploration of Common Duct in Conjunction with Cholecystectomy or Cholecystostomy**

Number of cases in which duct was explored	118
Percentage of total number of cases in which duct was explored	36%
Number of cases in which stones were found	44
Percentage of explored cases in which stones were found	37%
Percentage of total cases in which stones were found	13%
First five years:	
Number of cases in which duct was explored	37
Percentage of total number of cases in which duct was explored	25%
Number of cases in which stones were found	17
Percentage of explored cases in which stones were found	46%
Percentage of total cases in which stones were found	11%
Second five years:	
Number of cases in which duct was explored	81
Percentage of total number of cases in which duct was explored	44
Number of cases in which stones were found	27
Percentage of explored cases in which stones were found	33%
Percentage of total cases in which stones were found	15%

* Cases of secondary choledochostomy not included.

arteries. It was felt that the cerebral hemorrhage, demonstrated at autopsy, accounted for his clinical course and death.

EXPLORATION OF THE COMMON BILE DUCT

The data concerning exploration of the common bile duct is summarized in Table IV. Over the ten-year period the common bile duct was explored in 36 per cent of the patients, and stones were found in 37 per cent of the

explored cases, or 13 per cent of all patients operated upon for acute or chronic cholecystitis.

Comparative studies of the two five-year periods reveal an interesting trend. The incidence of exploration of the common bile duct increased from 25 per cent of the cases in the first five-year period to 44 per cent in the second five-year period. Stones were found in 11 per cent of all patients operated upon for acute or chronic cholecystitis (46 per cent of the ducts explored) in the first five-year period and in 15 per cent of all patients operated upon for acute or chronic cholecystitis (33 per cent of the ducts explored) in the second five-year period. Obviously, fewer patients were left with stones in the common bile duct during the second five-year period.

In this series of cases, exploration of the common bile duct did not increase the mortality; there was one death in the 118 cases of acute and chronic cholecystitis in which the duct was explored (0.8 per cent mortality), whereas the mortality in cases of acute and chronic cholecystitis without choledochostomy was 2.4 per cent, and the mortality for cases of chronic cholecystitis without choledochostomy was 1.9 per cent. The records are inadequate to determine whether or not choledochostomy increases morbidity. It is our impression that it does not, although these patients do have a hospital stay a few days longer than those subjected to simple cholecystectomy.

We have observed the generally accepted indications for exploration of the common bile duct. During the past two years we have routinely taken cholangiograms on the operating table. A Bucky diaphragm was placed beneath all patients who were subjected to biliary tract surgery. In the ordinary case, a ureteral catheter was introduced into the common bile duct through an incision made in the cystic duct. Ten cubic centimeters of Diodrast were injected through the catheter over a period of ten seconds. Films were exposed after 5 and after 10 cc. had been introduced. While this procedure was of only limited value, in more than the occasional case it identified stones in the common bile duct or a narrowing of the distal end of this duct. If choledochostomy was performed for an obvious indication, a cholangiogram was taken on the operating table after injecting Diodrast through the T-tube. This precaution, too, on a number of occasions revealed stones, often when the operator felt confident that all of them had been removed.

Postoperative cholangiograms identified stones remaining in the common bile duct of five patients. Undoubtedly, there were others who had residual stones, for routine postoperative cholangiograms have been taken only in the past three years.

Patients who exhibit a narrowing of the distal end of the common bile duct continue to be troublesome, and represent, for us, an unsolved problem. The recent suggestion of Mahorner[1] that the distal limb of the T-tube be brought through the sphincter of Oddi under transduodenal exposure is appealing.

There were 12 cases of secondary choledochostomy with no mortality. In three of these patients, rather large cystic duct stumps were removed. Two of

these patients have been relieved of their symptoms. More recently, we have partially denervated the common bile duct, as suggested by Womack and Crider,[2] in cases of secondary choledochostomy.

CARCINOMA OF THE GALL BLADDER

There were four carcinomas of the gallbladder in the series. One patient was found to be inoperable at surgical exploration and died at home a few months later. In another patient, who was jaundiced, an attempt to do a radical operation resulted in death from hemorrhage on the operating table. A third patient, in an early case in which the diagnosis was made by the pathalogist after operation, had a choledochostomy and later, when he was inoperable, a vitallium tube was implanted in the common bile duct. He lived nine months. The fourth patient was admitted to the medical service in a moribund state. Since surgical consultation was not requested, operation was not considered. An autopsy disclosed an advanced carcinoma of the gallbladder with extensive metastases.

All patients with carcinoma of the gallbladder had cholelithiasis.

Papillomas of the gallbladder were found incidentally in two cases of chronic cholecystitis and cholelithiasis.

DISCUSSION

A detailed discussion of preoperative care, operative technic, and postoperative care is not within the scope of this paper. Careful attention was given to fluid requirements, electrolyte and protein metabolism, and vitamin needs. Ambulation within 24 hours was practiced in the last three years. Complicating diseases were sought for and treated. Particular attention was given to postoperative vascular complications, of which there were many; in the past three years the majority of the patients have received anticoagulant therapy, prophylactic or therapeutic, or vein ligations.

With regard to anesthesia, one procedure, intercostal nerve block with procaine hydrochloride, has been of significant value. The efficacy of this anesthetic maneuver was first made apparent to us when it was used with satisfaction as the sole anesthetic agent for the repair of a ruptured peptic ulcer. Combined with inhalation anesthesia, as we commonly use it, it routinely afforded the muscular relaxation so necessary for obtaining proper exposure of the operative field. It reduced by approximately one third the amount of inhalation anesthesia required.

Technically, we have emphasized the use of careful dissection and fine cotton sutures. Drainage through a stab wound, now used routinely, was established in 62 per cent of the cases in this series.

Unhappily, the inadequacy of the records prior to the establishment of a residency, has rendered a study of morbidity invalid. The average highest postoperative temperature was 101.5 degrees F.

SUMMARY

A survey of biliary tract surgery performed in a small community hospital during a ten-year period ending May, 1948, has been presented. There were 348 operations with an overall mortality of 2 per cent.

In 85 cases of acute cholecystitis, 78 cholecystectomies, seven cholecystostomies, and 24 choledochostomies were performed with a mortality of 3.5 per cent. Patients with acute cholecystitis were regarded as emergencies; as soon as their metabolic disturbances were adequately corrected they were operated upon. Such a plan of treatment is particularly feasible for surgeons working in a small community hospital, since they fortunately see their patients early in their disease. The opportunity to perform cholecystectomy and choledochostomy in a high proportion of patients with acute cholecystitis, and yet maintain a reasonable mortality, is gratifying.

Cholecystectomy was performed in 246 cases of chronic cholecystitis with a mortality of 1.2 per cent. During the past five years, the mortality has been 0.7 per cent in 139 cases.

The common bile duct was explored in 36 per cent of the patients and stones were found in 13 per cent of all patients operated upon for acute or chronic cholecystitis. In the first five-year period, 25 per cent of the ducts were explored and stones were found in 11 per cent of all patients operated upon for acute and chronic cholecystitis. In the second five-year period, 44 per cent of the ducts were explored and stones were found in 15 per cent of all patients operated upon for acute or chronic cholecystitis. There was no increased mortality for common duct exploration. On the basis of this study, the higher incidence of common duct exploration seems justified.

Biliary tract surgery performed in a small community hospital does not compare unfavorably with that done in large metropolitan hospitals.

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ACUTE APPENDICITIS IN CHILDREN

A REVIEW OF ONE THOUSAND ONE HUNDRED AND SIXTY-FIVE CASES*

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SINCE THE APPARENT SOLVING of the riddle of appendicitis by the renowned American pathologist Reginald Fitz[1] in 1886, a considerable number of papers have been written which have considered the appendix under almost every conceivable condition.[2-9] Despite this, the mortality from a perforated appendix is still extremely high. This series is one of the larger reported in the literature and it is felt an analysis of our results should prove informative. The material for this study was taken from the Children's Surgical Service, Bellevue Hospital, and covers the period from 1926 to 1947 inclusive. This service accepts patients up to and including 12 years of age. The report does not cover all appendectomies performed but only those found on pathologic examination to fall in one of the following groups: (1) acute appendicitis unperforated, (2) abscesses, (3) spreading peritonitis. This classification is one that has been used by the senior author (P. D. A.) in previous published papers[10, 11] in which Groups A and B of this report were analyzed in detail. We have divided our patients into three groups (Table II). Group A covers the period from 1926 through 1935; Group B from 1936 through 1940, and Group C from 1941 through 1947. As a matter of convenience these groups will be referred to in the following text simply as Group A, B, or C.

CLASSIFICATION

Table I summarizes our results with the three pathologic groups previously defined. There were 708 cases (60.7 per cent) of acute appendicitis unperforated; 200 cases (17.1 per cent) with abscess, and 267 cases (14.3 per cent) with spreading peritonitis; 165 cases or 13.9 per cent occurred in children below the age of five years.

In Group A, 51 per cent were in the unperforated class; in Group B, 66.5 per cent; and in Group C, 72.6 per cent. This marked increase in the percentage of cases in the unperforated group in the past few years is visual evidence of the success of numerous public and private agencies in the work of educating the laity to recognize the signs and symptoms of appendicitis. In Group A there were 154 cases (25 per cent) falling in the abscess class; in Group B, 26 cases (8.8 per cent); and in Group C, 20 cases (7.6 per cent). It is evident from these statistics that there has been a steady decline in the percentage of cases admitted to the hospital with the diagnosis of appendiceal abscess. During the period between 1926 and 1935 there were 144 cases (24 per cent) of spreading peritonitis. This percentage remained the same between

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1936 and 1940, but dropped to 19.6 per cent during the period from 1941 through 1947.

There has been a progressive drop in the total number of patients admitted to this hospital in the past few years with the diagnosis of appendicitis. A number of explanatory factors have contributed to this, as we have no reason to believe that the incidence of appendicitis is decreasing. First there has been the creation of the "district system," whereby city hospitals are responsible only for well defined zones; secondly, because of the increasing wage scales and the increased use of voluntary health insurance programs, more people are seeking private hospitalization.[12]

MORTALITY AND FACTORS INFLUENCING MORTALITY

The overall mortality in our series of 1165 cases was 3.8 per cent. This contrasts favorably with Beekman's[13] reported mortality of 7.5 per cent in

TABLE I.—*Distribution and Death Rate of Patients According to Group.*

	Patients	Percentage	Deaths	Percentage Mortality
Unperforated	708	60.7	8	1.1
Abscess	200	17.1	5	2.5
Spreading peritonitis	267	14.8	31	11.6
Total	1165		44	3.8
Infants	165	13.9	15	9.09
Older children	1000	85.8	29	2.9

1924 on the same service. His report covers the period immediately previous to our Group A cases. There were eight deaths in the unperforated group, with a mortality of 1.1 per cent, five deaths in those with abscess with a mortality of 2.5 per cent; and 31 deaths in cases with spreading peritonitis, with a mortality of 11.6 per cent. There were only 29 operative deaths in this last group, permission for operation being refused in two instances (Table I).

In Group A there was a mortality of 2.2 per cent in the unperforated group. In Group B it had dropped to 0.51 per cent and in Group C there were no deaths. In Group A there was a mortality of 17.4 per cent among those patients with spreading peritonitis; in Group B the mortality had dropped to 4.2 per cent, while in Group C it was 5.8 per cent. The overall mortality in Group A was 5.8 per cent; in Group B, 1.6 per cent; and in Group C, 1.1 per cent. Table III compares our mortality with various other figures reported in the literature.

In the 165 patients under five years of age, there were 15 deaths, or 10 per cent. Beekman,[13] in 1924 reported a mortality in this group of 25.6 per cent on the same service. The high death rate from spreading peritonitis in the infant is well known. The reasons for this high mortality are still not completely explained, but the following factors are thought to play an important role: (1) the increased time elapsing from onset of symptoms to operation as

compared to the older age groups because of greater difficulty in making the diagnosis, (2) the inability of the underdeveloped omentum to wall off perforations, and (3) the greater difficulty in establishing and maintaining fluid, protein and electrolyte balance in the young infant.

TABLE II.—*Division of the Total Cases into Three Groups Showing Distribution and Death Rate.*

	Patients	Percentage	Deaths	Percentage Mortality	
Group A—1926–1935					
Unperforated	314	51	7	2.2	In the 72 cases under five
Abscess	154	25	4	2.5	years of age, there were 11
Spreading peritonitis	144	24	25	17.4	deaths. Of these, 9 were in
Total	612	100	36	5.8	the spreading peritonitis
Infants	72	11.7	11	15.27	class (81 per cent).
Other children	540	88.3	25	3.9	
Group B—1936–1940					
Unperforated	195	66.5	1	0.51	In the 50 cases under five
Abscess	26	8.8	1	3.8	years of age, there was one
Spreading peritonitis	72	24.5	3	4.2	death. Permit for opera-
Total	293	100	5	1.6	tion denied. Autopsy
Infants	50	16.1	1	2.0	showed spreading perito-
Older children	243	82.9	4	1.6	nitis.
Group C—1941–1947					
Unperforated	189	72.6	0	0.0	In the 43 cases under five
Abscess	20	7.6	0	0.0	year of age there were 3
Spreading peritonitis	51	19.6	3	5.8	deaths, all in the spreading
Total	260	100	3	1.1	peritonitis group.
Infants	43	16.5	3	6.9	
Older children	217	83.4	0	0.0	

TABLE III.—*The Reported Mortality of Appendectomy.*

Author	Patients	Percentage Mortality
Beekman	145	7.6
Maes	250	7.6
Finney	193	6.2
Woodall	295	7.45
Caldwell	220	2.7
Ladd & Gross	940	3.1
Abel & Allen	1165	3.8
Keyes	263	11.0
Tasche	82	8.5
Penberthy, Benson & Weller	1653	4.2

In addition to the aforementioned factors, there are other variables which must be considered to influence mortality, viz.: (a) hours from symptom onset until admission, (b) hours from admission until operation, (c) whether catharsis has been given, (d) age of the patient, (e) severity of pathologic changes in the appendix, (f) whether chemotherapy or antibiotics are administered, and (g) the surgical technic employed.

A. *Time from Symptom Onset until Admission to Hospital.* This time has gradually been reduced from an average of 49.1 hours in Group A, to 36.2 hours in Group C. The cases of appendicitis with abscess formation had the

longest time from symptom onset until admission. In Group A this averaged 147 hours; in Group B, 88 hours and in Group C, 87 hours. From the figures in Table I, it is evident that the highest mortality lay in the spreading peritonitis group. It has been shown[14] that the incidence of appendicitis with perforation begins to climb steeply after the first 24 hours. It follows from this then that it is imperative to make an early diagnosis and to arrange for early hospitalization of the patient. This responsibility rests for the most part with the pediatrician and family doctor and it is incumbent upon them to realize the importance of early diagnosis and to enlist the early aid of a competent surgeon.

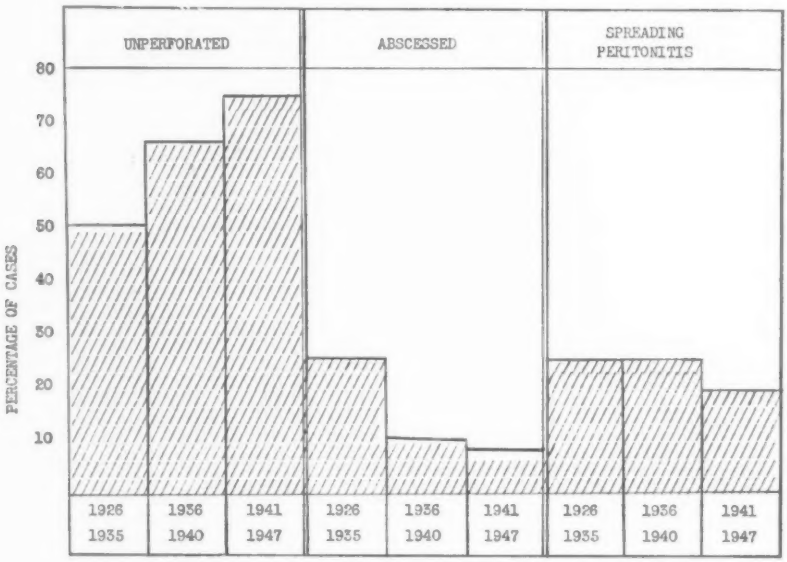

FIG. 1.—Relative severity of disease over a period of 22 years.

B. *Hours from Admission to Operation.* In Group A this averaged 3.9 hours; in Group B, 7.5 hours and in Group C, 9.1 hours. This upward trend does not indicate that we believe acute appendicitis is not a surgical emergency, but rather that ample time is being allowed for the correction of any existing fluid or electrolyte imbalance. In those cases where this is satisfactory on admission, immediate operation is the procedure of choice. This keener awareness of the importance of preoperative preparation has been an instrumental factor in reducing the mortality and postoperative complications. This is borne out, we think, in the relatively smooth postoperative course of these patients in the past few years.

C. *Catharsis.* The factor of catharsis has definitely shown a trend in the right direction. In Group A, 49 per cent gave a history of catharsis; in Group B, it dropped to 36 per cent and in Group C to 19.6 per cent. Enough statistical evidence has been accumulated showing that purgatives increase intestinal

peristalsis thereby potentially increasing the chance of perforation, which warrants continued education of the laity regarding this danger.

D. *Age.* Of the total number of cases, 14 per cent were under the age of five years. There were 15 deaths in this group with a mortality of 10 per cent compared to 3 per cent for those over five years. Of the 15 deaths, 13 (86.6 per cent) were in the spreading peritonitis group. Figure 2 reveals that with increasing age there is a marked and steady decline in the mortality. However, the age, *per se,* is not the only factor which determines the high mortality in those under five years.

E. *Degree of Inflammation.* Obviously this plays an important role in helping to determine the mortality. In the unperforated group the mortality was 1.1 per cent. With abscess formation, 2.5 per cent, and when spreading peritonitis was present it rose sharply to 11.6 per cent.

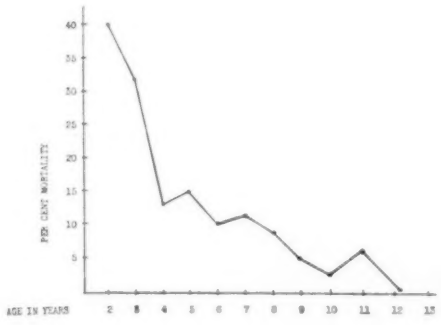

FIG. 2

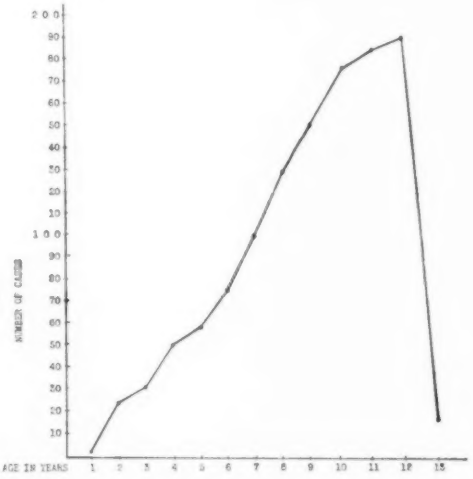

FIG. 3

FIG. 2.—Mortality rate in percentages according to year of age.
FIG. 3.—Incidence of appendicitis as the child grows older.

F. *Antibacterial Agents.* An important factor in the reduction of the morbidity and, to a lesser extent the mortality, has been the discovery and mass production of antibacterial drugs. It should be remembered that the mortality from appendicitis had begun to drop before the antibacterial drugs were freely available, and other factors, such as (1) early diagnosis, (2) free use of whole blood when necessary, (3) use of parenteral fluids, (4) Levin tube for intragastric suction, (5) postoperative oxygen prophylactically, and (6) the use of the McBurney incision, should not be forgotten as having played an important role in this drop. There have been no well defined rules on this service regarding the use of the sulfonamides either locally or parenterally; rather it has been left to the individual surgeon to decide on the basis of his operative findings. Numerous articles[21-31] have appeared in the liter-

ature which deal exhaustively with this subject. The use of peniciillin in connection with spreading peritonitis is discussed later.

G. *Incision.* From 1926 through 1935, 95 per cent of all patients were operated upon through a right rectus incision, and 5 per cent through a McBurney incision (mortality 5.8 per cent). During the period from 1936 through 1940, 93 per cent were done through a McBurney, and 7 per cent through a right rectus incision (mortality 1.6 per cent). From 1941 through 1947, 96 per cent were done through a McBurney, and 4 per cent through a right rectus incision (mortality 1.1 per cent). Meyer, Requarth and Kayall[15] report that "a McBurney incision is associated with the lowest mortality; a right rectus was higher and the midline incision had the highest of all. The closer to the midline the incision is made, the higher will be the mortality."

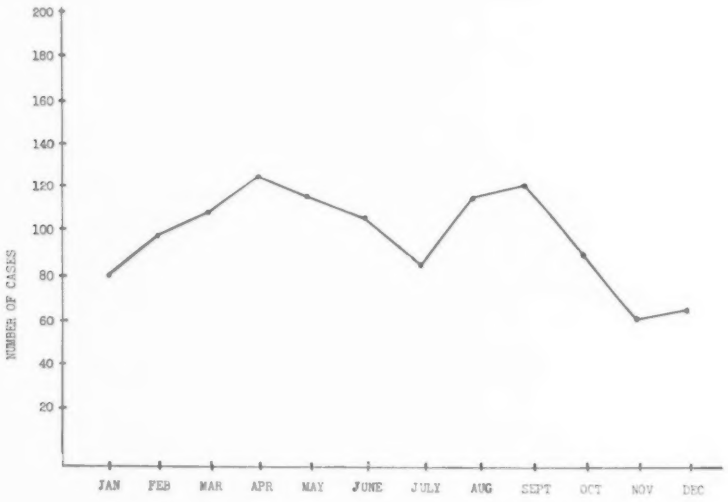

FIG. 4.—Monthly incidence of 1165 cases.

AGE, COLOR AND SEX INCIDENCE

Appendicitis is rare under one year of age. There were only two cases in our series in the one year old group (0.17 per cent). It is uncommon during the second year, but thereafter (see Fig. 3) increases as the child grows older.

In 1165 cases there were 35 (3 per cent) in the colored race. The incidence is slightly higher in males, being 59 per cent to 41 per cent in females. This is approximately a 2:1 ratio, which agrees substantially with other reports in the literature.[12, 14]

SEASON

Though attempts have been made to correlate the seasonal and monthly incidence, the results are not very significant. There was a definite rise from

January through April, followed by a slight drop and then a rise again to a peak in August. Thereafter the incidence dropped sharply (Fig. 4). It has been suggested that this higher incidence in the spring is possibly related to the increased incidence of respiratory infections during this period.

TREATMENT

Once the diagnosis of acute appendicitis has been made, early operation is the only justifiable procedure. To delay longer than is necessary in order to restore hydration and electrolyte balance invites disaster. It is well to remember that the degree of infectious process is difficult to determine clinically and perforation may be imminent at any time; as pointed out before, the mortality and morbidity rise sharply following perforation.

The decision regarding the choice of an anesthetic agent rests with the individual preference; in this series open drop ether or cyclopropane was used. In the final analysis we feel it is the anesthetist more than the anesthesia that determines whether there is a smooth induction and whether there will be post-anesthetic complications. We are fortunate in that all anesthesia here is given by doctor-anesthetists.

It has been mentioned previously that from 1941 through 1947, 96 per cent of our cases were done through a McBurney incision. In addition to the well substantiated fact[15] that a McBurney incision is associated with the lowest mortality, there are other factors which indicate that this incision is the one of choice. Briefly they are: (1) The operative procedure may be carried out with a minimum of peritoneal contamination; (2) there is less danger of injury to nerves and blood vessels than in vertical incision; (3) the incision gives the best exposure for the expected abnormal findings; (4) if the appendix is retrocecal, lateral extension of the incision will expose the lateral posterior wall of the cecum whereas upward extension of a right rectus would tend to lie medial to the site of the appendix;[15] (5) if the appendix is medial, further exposure may be obtained by means of Wier's extension; and (6) this incision is associated with a lower incidence of postoperative hernia.

It has been customary on this service to drain appendiceal abscesses once the diagnosis has been made. The incision is made over the mass, and the peritoneal cavity entered with great gentleness so as not to disseminate the infection. The object of the surgeon should be to establish drainage with the least possible manipulation. Suitable drains are inserted and no attempt is made to remove the appendix unless it presents itself so that it may be removed without disturbing the architecture of the abscess wall. These patients should be advised that the appendix still remains and re-admitted two to three months later for elective appendectomy.

In those cases of spreading peritonitis, we feel that early operation is the procedure of choice. Adequate time is allowed, however, for restoration of normal fluid balance. Drains are inserted and the peritoneum closed around the drains. In recent years we have used drainage less frequently. Many

patients with early spreading peritonitis are not drained, but after closing the peritoneum the rest of the abdominal wall is left open. The wound is loosely packed open with petrolatumed gauze and dressed in 48 hours. Penicillin is started parenterally preoperatively and continued until no longer indicated. In addition, depending upon the preference of the surgeon, sodium sulfadiazine may be given in the infusion during the operation. Postoperatively, in addition to the usual supportive measures, oxygen is given when indicated and intragastric suction as needed.

Since the introduction of penicillin, numerous reports[17-20] have appeared in the literature discussing its use in various degrees of appendicitis. Ochsner and Johnston[17] report a series of 50 cases of spreading peritonitis treated with penicillin in which there was but one death. All masses resolved. In no case was it necessary to drain an intra-abdominal abscess and there was no spontaneous drainage into the bowel. They also estimate that in cases of appendiceal peritonitis treated conservatively, three-fourths will subside spontaneously without going on to abscess formation. In the remaining 25 per cent, incision and drainage of the abscess will be necessary. There is no question that penicillin is a valuable adjunct in the treatment of appendicitis, but we feel that it should remain as an adjunct only to competent surgery.

Management of the appendiceal stump varies with the operating surgeon. Generally, however, we favor tying the stump with a very fine plain catgut ligature and then inverting this ligated stump with a single fine chromicized catgut purse-string suture. We believe the argument that we are inverting an infected stump into a blind pocket is fallacious. If pus should accumulate here it will most certainly rupture into the bowel. This tie provides insurance against hemorrhage into the bowel. In those patients where there is an extension of the edema and inflammation on the cecal wall, we do not attempt to invert, but doubly ligate the stump with two chromicized catgut ties. We usually amputate the appendix with the carbolized knife and then apply alcohol to the stump.

The postoperative care of these patients has been discussed concomitantly with the presentation of other data and will not be taken up as a separate topic. There is one important point that warrants mentioning, however, and that is the extreme desirability of early co-operation with the pediatrician to forestall, or at least to be more adequately prepared for, any medical emergencies that might arise.

DAYS IN HOSPITAL

The number of hospital days has gradually been reduced from an average of 21 days in Group A, to 10.3 days in Group C. Many factors have undoubtedly contributed and have been mentioned previously in this paper. The two most important ones are, first, the increasing number of cases in the unperforated class; and secondly, the more general use of the McBurney incision.

SUMMARY

1. A review of appendicitis in children from a large city hospital covering a 21-year period has been presented.

2. The overall mortality has dropped from 5.8 per cent in Group A, to 1.1 per cent in Group C.

3. The mortality in those cases of spreading peritonitis remains high. Some of the factors deemed responsible have been discussed.

4. The total hospital days have been reduced and the contributing factors have been presented.

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ACUTE PANCREATITIS AND DIABETES*

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ONE OF THE LESS COMMON but more arresting complications of acute pancreatitis is diabetes mellitus. This combination of diseases is either exceedingly rare or its presence is frequently overlooked, for surgical texts and current medical literature devote remarkably little attention to the subject. A case of acute pancreatitis complicated by the appearance of diabetes has stimulated a critical review of our past records with respect to this phenomenon. The results of this study have led us to conclusions contrary to those recorded in the literature and are, for that reason, worthy of comment.

CASE REPORTS

Case 1.—A white man, 37 years of age, was admitted to the Henry Ford Hospital August 18, 1943, complaining of excruciating epigastric pain. It began mildly during the preceding night, following 4 or 5 drinks, and became suddenly severe just before noon. The pain was not relieved by morphine and was extreme on admission, approximately 3 hours after the onset of the acute attack. It extended across the entire upper part of the abdomen but was most severe in the epigastrium. Vomiting occurred but was not extreme.

The past and family history was entirely devoid of diabetic symptoms. The patient had suffered from recurrent attacks of upper abdominal distress which usually followed the ingestion of large meals. Recent cholecystograms, taken elsewhere, showed numerous stones in the gallbladder.

The patient was moderately obese and obviously in great pain, pale and perspiring. The abdomen was rigid in the epigastrium but soft in the lower segments. Tenderness was present to some degree over the entire abdomen but was most exquisite in the epigastrium and below the right costal margin. Rebound phenomenon was present in the upper part of the abdomen. The temperature was 100.2° F., pulse 84, respirations 22 and blood pressure 128 mm. systolic and 78 mm. diastolic. Laboratory data revealed 17.0 Gm. of hemoglobin per 100 cc., leukocytes numbered 17,000, blood sugar 198 mg. per 100 cc., and blood diastase (Myers) 76. Urinalysis was negative. A roentgenogram of the abdomen showed no free air in the peritoneal cavity.

A tentative diagnosis of acute pancreatitis was made and operation was withheld as is our custom when the diagnosis is reasonably certain. Continuous gastric suction was instituted and parenteral fluids, including 600 cc. of plasma, were administered. After 5 hours the degree of hemoconcentration was unaltered but the diastase had risen to 91.

Severe abdominal pain and diffuse tenderness persisted during the first 3 days of the illness and were accompanied by marked ileus. The blood sugar the morning of the

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fourth day was 268 mg. per 100 cc. That afternoon the patient became listless and his respirations became Kussmaul in character. Urinalysis revealed 4 plus acetone and diacetic acid; the blood plasma carbon dioxide combining power was 25 volumes per 100 cc. The patient was treated vigorously for diabetic acidosis, with favorable response. The severity of the diabetes is indicated by the data contained in Figure 1 which reveal the daily variations in blood sugar, carbon dioxide combining power and insulin requirements. Figure 2 shows the fluctuations in the leukocyte count, blood diastase and hemoglobin values.

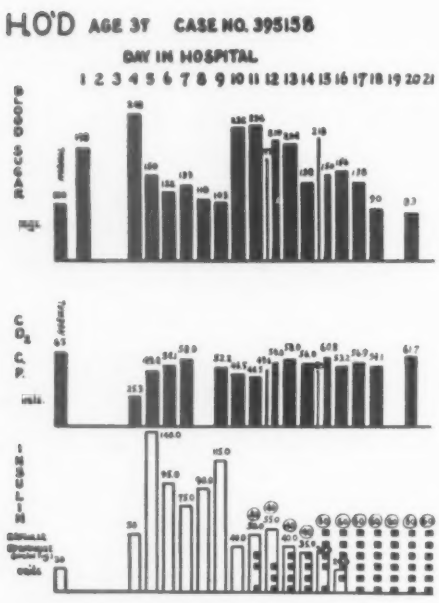

FIG. 1

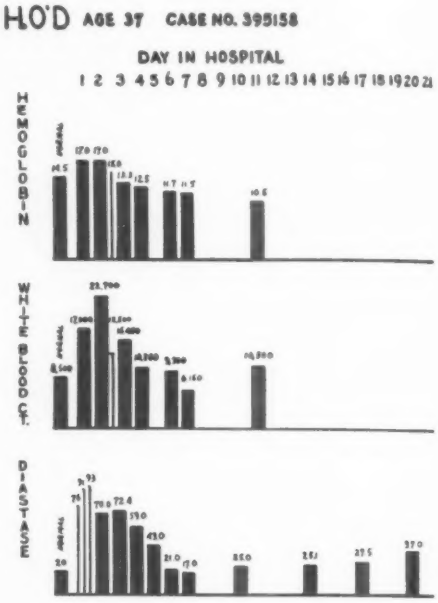

FIG. 2

FIG. 1.—Daily variations in blood sugar, carbon dioxide combining power and insulin requirement during the first three weeks of illness are represented graphically. Diabetic acidosis was evident on the fourth day of illness. Large amounts of regular insulin were required to control the diabetes. Later, the glucose metabolism was regulated with 60 units of protamine zinc insulin daily.

FIG. 2.—The hemoconcentration early in the disease is reflected in the increased hemoglobin values. Anemia develops later as the course of the disease continues. Initial leukocytosis accompanies the acute phase of the disease. Diastatic activity of the serum is greatly increased during the first 72 hours and then returns to normal.

On the fifth day of the illness the diagnosis of acute pancreatitis was confirmed by the presence of a tender, elongated mass across the upper part of the abdomen. The pain, which was constant in the beginning, became intermittent. Gradually the marked toxicity abated, intestinal activity returned, and the insulin requirement diminished, but the swollen, tender mass remained.

The patient was discharged on September 20, 1943, to await a more propitious time for cholecystectomy. He was re-admitted on October 5, 1943. During the interim he continued to have moderately severe pain, particularly after meals, but the pancreatic mass was smaller and less tender.

At operation, October 8, 1943, the abdomen was opened through a Kocher incision under Nupercaine anesthesia. Exploration revealed extensive fat necrosis throughout the

peritoneal cavity. The pancreas was greatly enlarged and extremely nodular with an area of softening near the tail. The gallbladder contained many stones. The common bile duct, which was not dilated, was explored and no stones were found. The gallbladder was removed and the common duct was drained. The postoperative course was stormy. On the tenth postoperative day a spiking temperature developed, associated with periodic chills, which persisted during the remainder of the illness. The patient died November 9, 1943.

Autopsy revealed the pancreas to be completely necrotic, with an abscess cavity occupying the entire pancreatic area. The common duct, which was not dilated, contained one small gallstone. The areas of fat necrosis were partially calcified.

Case 2.—A white woman, 38 years of age, was admitted to the Henry Ford Hospital August 12, 1941, complaining of severe epigastric pain which had come on suddenly 12 hours before admission. Nausea and vomiting accompanied the pain. The patient was acutely ill. There was extreme tenderness in the epigastrium and right upper quadrant. There was a large, firm, irregular mass in the lower abdomen extending to the level of the umbilicus. Urinalysis showed no sugar and no albumin. Blood diastase (Myers) determination made on a specimen obtained approximately 24 hours after the onset of pain was 39.7. The hemoglobin was 16.3 Gm. per 100 cc., erythrocytes numbered 5,400,000 and leukocytes 20,000, with 88 per cent polymorphonuclear leukocytes. On the second day following admission urinalysis disclosed 4 plus sugar and 4 plus acetone. In a note on the patient's record, made by the intern, the possibility was considered of diabetes complicating pancreatitis in this instance. Three days after the initial symptoms, a firm, tender mass was palpable in the epigastrium. Two days later the blood sugar was 184 mg. per 100 cc. The clinical course gradually improved. The patient was discharged from the hospital on August 26, 1941.

Cholecystograms one week later showed no visualization of the gallbladder. In November, 1941, the patient had a hysterectomy. At operation, the gallbladder was of normal thickness and devoid of stones. Several irregular nodules were present in the region of the pancreas. In the upper abdomen, just to the left of the mid-line, there was a large mass made up of many smaller nodules. On the left side, overlying the left kidney, there was a similar nodular mass. One of the nodules was excised for microscopic examination and revealed fat necrosis and fibrosis. Following the operation the patient complained of severe pruritus. The blood sugar on December 15, 1941, was 282 mg. per 100 cc. The glucose tolerance curve confirmed the diagnosis of diabetes. The diabetes was controlled with insulin for two months and is now managed by dietary restrictions. Glucose tolerance is still abnormal.

Case 3.—A man, 37 years of age, was admitted to the Henry Ford Hospital November 9, 1941, complaining of pain in the left upper portion of the abdomen and left lower chest, of 2 days' duration. The pain was accompanied by persistent vomiting. The acute illness followed a heavy and protracted drinking spree. The pain was sharp and persistent and extended through the left chest to the tip of the scapula. A moderate fever and productive cough had been present since the second day of the illness. The patient had some frequency and dysuria. The past history was dominated by chronic alcoholism with periodic excesses. The patient had had 3 previous attacks of dull pain in the right upper quadrant of the abdomen. There was no history of jaundice, alteration in bowel habit or stool. There was no familial or personal history of diabetes.

Examination on admission revealed that the patient was acutely ill and moderately obese. The temperature was 102° F., pulse rate 130 and respirations 48. He was dyspneic but not cyanotic. Examination of the chest disclosed the diaphragm to be high on each side. The percussion note was dull over the left base posteriorly. The breath sounds were tubular and there were numerous râles in this area. The blood pressure was 110 mm. systolic and 80 mm. diastolic. There was tenderness to fist percussion in the left costovertebral angle. The abdomen was tensely distended and was uniformly tender. Rebound phenomenon was present in the left upper quadrant. No abdominal masses were palpable.

Initial laboratory data showed the hemoglobin to be 13.5 Gm. per 100 cc. of blood, erythrocytes numbered 4,480,000, leukocytes 7600, with 85 per cent polymorphonuclear leukocytes. Urinalysis revealed 2 plus sugar, but no acetone or diacetic acid. An occasional white blood cell and a few hyaline and granular casts were present. The stool was tarry and the guaiac test was 3 plus. Type 16 pneumococci were demonstrated in the sputum. The blood sugar was 382 mg. per 100 cc., and the carbon dioxide combining power was 25.3. Blood diastase was not determined until the fifth hospital day, at which time it was 14.3 mg. per 100 cc.

Abdominal pain and distention persisted while the area of pulmonary dullness subsided under sulfadiazine therapy. The leukocyte count rose steadily to 21,000.

Five days after admission a mass could be felt in the left flank. The temperature varied between 100 and 103° F. An intravenous pyelogram on the eighth day of hospitalization showed no abnormality.

A diagnosis of perinephric abscess was made and surgical drainage was planned. Examination of the abdomen following pre-anesthetic medication revealed a separate mass in the upper abdomen just to the left of the midline. Roentgenograms of the stomach following a barium meal showed a large deformity of the greater curvature resulting from an extrinsic mass. Two days later, incision and drainage of the mass in the left flank was done through the usual kidney incision and a thick-walled cyst not involving the kidney was found. The anterior mass was not affected by the procedure so the patient was placed in the supine position and the abdomen was opened through an upper midline incision. A large cyst arising in the pancreas and filling the lesser peritoneal sac was drained of 800 cc. of dark red fluid. The diastatic activity of this fluid was 62. The cyst was marsupialized.

Convalescence was slow but satisfactory. The patient was discharged from the hospital 9 weeks after operation, at which time the incision in the left flank was healed. The epigastric wound drained for 3 more weeks. The diabetes, which was present upon admission, persisted following recovery and was controlled by diet and small doses of insulin.

Case 4.—A man, 52 years of age, was admitted to the emergency room March 23, 1944, at 1 P.M., complaining of severe epigastric pain of 4 hours' duration. The pain was accompanied by considerable vomiting. In recent years he had had several similar attacks, but these were less severe and of short duration.

The patient was well nourished but appeared acutely ill. The temperature was 98.8° F., the pulse 120 and respirations 28. The abdomen was soft except for slight splinting in the epigastric region. The leukocyte count was 20,000. The blood diastase (Myers) was 54. The urine contained no sugar.

A diagnosis was made of acute pancreatitis and conservative treatment, including continuous gastric suction, parenteral fluids and oxygen, was instituted. The following morning the clinical appearance was unchanged. The urine contained 4 plus sugar and 1 plus acetone. The blood sugar was 250 mg. per 100 cc. and the carbon dioxide combining power was 34.5. He was placed on 10 units of regular insulin every 2 hours until the urinary sugar was 1 plus or less. The insulin requirement varied from 30 to 50 units per day from the onset of diabetic manifestations to the termination of the illness.

The abdomen became progressively distended and a faint purplish discoloration appeared in each flank on the third day of the illness (Gray-Turner sign). The clinical course was one of gradual deterioration despite adequate control of the diabetic state and vigorous supportive measures. Death occurred on the fifth day of the illness.

Postmortem examination revealed extensive fat necrosis in the omentum and mesentery. There was an area of necrosis in the right iliacus muscle. The gallbladder contained stones. There was extensive hemorrhagic necrosis of the body and tail of the pancreas. The duct of Wirsung could not be identified. The head of the pancreas was less severely involved in the necrotizing process, but numerous areas of destruction were present.

Microscopic study showed only an occasional island of Langerhans in the head of the pancreas. No islets were observed in the body or tail of the gland.

Case 5.—A man, 34 years of age, was seen in the Henry Ford Hospital for routine physical examination. A fasting blood specimen revealed normal sugar concentration. A diagnosis of chronic alcoholism was made. He was next seen on August 9, 1943, following a long bout of alcoholic excess. Two weeks before this admission, following a heavy meal, he had a sudden onset of severe epigastric pain, which was constant and nonradiating.

The patient was thin and nervous. Moderate tenderness and rigidity were present in the epigastrium. The fasting blood sugar was 130 mg. per 100 cc. and the diastase (Myers) was 18. Roentgenologic examination of the abdomen revealed diffuse areas of increased density in the upper part of the abdomen which were interpreted as pancreatic calculi.

Two days after this admission the patient had a recurrence of the severe epigastric pain. The upper abdomen was firm and exquisitely tender. There was an indefinitely outlined mass beneath the area of tenderness. The leukocyte count was 18,000. A diagnosis of recurrent acute pancreatitis was made and nonoperative supportive measures were instituted. The pain and the abdominal mass gradually subsided and the patient was discharged on August 25, 1943. Roentgenologic examination following a barium meal demonstrated a widened duodenal sweep, indicative of enlargement of the head of the pancreas. Multiple pancreatic calculi were demonstrated in the same film.

A fasting blood sugar estimation on September 11, 1943, gave a value of 210 mg. per 100 cc. and a glucose tolerance test on October 4, 1943, was characteristic of diabetes mellitus. The patient suffered from polyuria, polydipsia and recurrent epigastric pain. Administration of protamine insulin, 20 units per day, effectively controlled the diabetic symptoms and the hyperglycemia.

The patient was re-admitted to the hospital on July 23, 1944, because of recurrent epigastric pain. The abdominal findings were unchanged. The diabetic state was adequately controlled by diet and 10 units of protamine insulin. Pancreatic resection was advised for the relief of the pain but the patient declined. He was discharged from the hospital on July 27, 1944, and no further observations have been made.

LITERATURE

Harley,[1] in 1862, reported a case of acute pancreatitis exhibiting glycosuria and Atkinson,[2] in 1895, and Körte,[3] in 1911, described several similar cases. Fitz,[4] curiously enough, made no mention of diabetes in his original contributions to the literature of acute pancreatitis, but Shumacker,[5] in 1940, estimated that 11 per cent of patients with acute pancreatitis had glycosuria during some phase of the acute attack.

If glycosuria is a relatively rare finding in acute pancreatitis, hyperglycemia is not. Brocq and Varangot[6] collected 72 cases of acute pancreatitis in which blood sugar determinations were made and found that in 15 instances it was below 150 mg. per 100 cc.; in 23 it was between 150 and 200 mg., and in 34 cases it was over 200 mg. The frequent occurrence of hyperglycemia was recognized by Douglas[7] and by Cole,[8] but only the latter attributed to it diagnostic significance.

Several authors, including Bernhard,[9] Mikkelsen[10] and Wildegans,[11] believe that alterations in glucose tolerance are more common and of greater significance than is either glycosuria or hyperglycemia, because disturbances in glucose tolerance may occur in the milder cases of acute pancreatitis and are much less likely to be observed in other acute abdominal conditions which are easily

confused with pancreatitis. For this reason it is thought that the glucose tolerance test should be used with increasing frequency both during the acute attack and in the postconvalescent period.

Generally speaking, disturbances in carbohydrate metabolism related to pancreatitis are transient, but several instances of true diabetes, resulting from acute pancreatitis, have been recorded. Shumacker,[5] in his comprehensive review, found 62 cases of diabetes which he believed were caused by acute pancreatitis and added one of his own, but in only 25 of the collected cases did the diabetes appear concurrently with the acute attack. Of these 25 patients, six died of diabetic acidosis shortly after operation and 13 died of other causes during the acute illness. Two of the six survivors died within a short time of diabetic acidosis. Shumacker's patient survived but had persistent diabetes. Four of the remaining 37 patients developed diabetes shortly after the acute attack. In the other 33 cases the causal relationship between acute pancreatitis and the subsequent diabetes is presumptive for the diabetes developed from one to 20 years after the acute attack. In 1942, Rodney Smith[12] added to the literature another fatal case of acute pancreatitis complicated by diabetes.

In some instances disturbances in glucose metabolism, and even diabetes, have been reported in other pancreatic lesions, such as pseudocysts, chronic pancreatitis, traumatic calcification and extensive carcinoma, but the causal relationship in such instances was less obvious than in the cases of acute pancreatitis. It is the consensus among investigators that when diabetes resulting from acute pancreatitis occurs, it is found in those instances in which the inflammatory process has been severe, the extent of necrosis great and the period of sequestration prolonged.

Although acute pancreatitis complicated by diabetes carries a high mortality, pre-existing diabetes complicated by acute pancreatitis is more deadly still. Shumacker stated that of 19 recorded cases only one patient survived. Recently Steiner and Tracy[13] reported the case of a child who died in diabetic coma and who had unsuspected acute pancreatitis and *B. welchii* peritonitis at autopsy. Most authors agree that acute pancreatitis occurring in the known diabetic is extremely serious, and that the presence of the abdominal complication is always difficult to detect.

COMMENT

In an effort to study the problem of the relationship of acute pancreatitis to diabetes we have analyzed the case records of 72 patients seen in the Henry Ford Hospital between 1917 and July 1, 1944, in whom the diagnosis of acute pancreatitis was established. The data with respect to the status of glucose metabolism in these records were inadequate in the majority of cases owing to the fact that proper attention has not been given to this feature of the disease, but it is a striking and surprising fact, nevertheless, that in this small series in which 38 cases were classified as severe, there were five cases of acute pancreatitis complicated by clinical diabetes. These figures appear even more significant when one realizes that in 12 other patients who died of the acute

pancreatitis, no information is available upon which to establish the presence or absence of diabetes as a complication of the primary disease. These five patients with diabetes were observed during a period of four years, from 1941 to 1944.

Although the cumulative literature on this subject indicates that diabetes complicating acute pancreatitis is a rare phenomenon, a careful examination of the individual reports suggests that adequate information regarding disturbances in glucose metabolism during attacks of acute pancreatitis is not available in any single series to warrant dogmatic conclusions as to the true incidence of this complication. We believe that our studies indicate that diabetes as a complication of acute pancreatitis is not a rare phenomenon and that, indeed, it may be reasonably common in that small group of patients who exhibit the devastating type of the disease characterized pathologically by extensive hemorrhagic necrosis. It is precisely this variety of acute pancreatitis which accounts for the majority of the deaths from the disease, and therefore any new facts which we may discover regarding the character of this condition may alter the mode of treatment and improve the prognosis.

It has been the practice of the various authors to explain the apparent rarity of diabetes complicating acute pancreatitis by assuming that in instances of gross pancreatic disruption of sufficient severity to produce diabetes the pancreatitis would be rapidly fatal and the patient would not live long enough to develop clinical diabetes. This may be true, but it is not possible, on the other hand, that in such rapidly fatal instances the victims have an acute, unrecognized insulin insufficiency which contributes to the mortality? Is it not plausible that the necrotic hemorrhagic pancreas commonly seen at autopsy in patients exhibiting the hyperacute form of the disease is incapable of adequate insulin secretion? These theoretical possibilities are consistent with the common observation that diabetes occurring in the course of acute pancreatitis is seen in those instances in which the degree of pancreatic necrosis has been extreme. The final answer to the question of the true incidence of diabetes complicating acute pancreatitis, and to the more important consideration of its contribution to the mortality of the primary disease, can be determined only by a careful study of glucose metabolism during all attacks of acute pancreatitis, and at appropriate intervals thereafter in a sufficiently large series to be statistically significant.

Assuming that diabetes complicating acute pancreatitis is more common in the fulminating types of the disease than has been suspected heretofore, is it possible that the excessive mortality of early operative intervention in these cases is due, in part, to associated unsuspected alterations in glucose metabolism? Since these alterations will occur almost exclusively in those instances in which the degree of pancreatic destruction is extreme, the mortality in this group of patients will continue to be high; nevertheless, it seems advisable to determine the exact status of carbohydrate metabolism before surgical intervention, and to follow it carefully during the postoperative period, particularly

if early operation is to be carried out. We feel that one of our patients (Case 1) might have survived had we delayed operation longer.

Finally, is it possible that undetected acute pancreatitis occasionally accounts for the abdominal pain of diabetic acidosis and that the presence of this complication influences the outcome adversely? In this regard it is noteworthy that in 19 cases of pancreatitis occurring in previous diabetics (Shumacker), the detection of the pancreatitis was very difficult. In an autopsy survey of 26 patients who died in diabetic acidosis, Root[14] reported four cases of acute pancreatitis complicating diabetic coma, all of which were unrecognized clinically. In the case reported by Steiner and Tracy the presence of pancreatitis was not detected prior to postmortem examination. This meager information suggests that the possible presence of acute pancreatitis should be carefully considered when patients complain of abdominal pain during episodes of diabetic acidosis.

CONCLUSIONS

Five cases of acute pancreatitis complicated by diabetes are described.

Alterations in carbohydrate metabolism related to pancreatic lesions are reviewed.

The possibility is raised that undetected disturbances in glucose metabolism existing before and after the operation may contribute to the very high mortality of the early operative treatment of acute pancreatitis.

A plea is made for thorough evaluation of carbohydrate metabolism in all patients suffering from acute pancreatitis.

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RESTORATION OF FUNCTION OF THE SHOULDER FOLLOWING PARALYSIS OF THE TRAPEZIUS BY FASCIAL SLING FIXATION AND TRANSPLANTATION OF THE LEVATOR SCAPULAE*

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This article is companion to that of Dr. K. G. McKenzie and Dr. Eben Alexander, Jr.—"Restoration of Facial Function by Nerve Anastomosis" which appeared in this journal September, 1950. In it they advise the use of the hypoglossal rather than the spinal accessory nerve as a motor for the facial muscles because denervation of the trapezius muscle by section of the spinal accessory nerve results in disabling drop-shoulder in some cases. This article recounts the complaints of such a patient, and describes an operation designed to compensate for the loss of function of the trapezius muscle. The patient's disability was almost entirely relieved by redistribution of the forces acting on the scapula.

Mrs. B. was a farmer's wife and accustomed to the usual heavy chores. She was right-handed. Skillful extirpation of an acoustic neuroma left a disfiguring right-sided facial palsy that was partially relieved by anastomosis of the spinal accessory to the facial nerve. This operation paralysed the trapezius muscle. She developed a severe drop-shoulder, even at rest (Fig. 1) and inability to abduct her arm more than 90 degrees (Fig. 2). Forward flexion was similarly limited. Farm duties became a burden. When carrying a pail of water it hung so closely to the right leg that walking was difficult. Paraesthesias occurred in the arm, forearm, and hand, due to traction on the brachial plexus—though examination failed to reveal any weakness or wasting. Abduction produced discomfort in the scapulohumeral joint. Passively the range of motion of the shoulder was unimpaired.

The trapezius and sternocleidomastoid muscles were devoid of activity. No surviving muscle fibers were found in the trapezius when it was exposed later. The profile of a much hypertrophied and powerful levator scapulae muscle stood out in the deepened hollow of the neck (Fig. 1). In the resting position the unopposed weight of the arm rotated the scapula on the chest wall, depressing the glenoid. When the shoulder was shrugged or the arm abducted the levator scapulae pulled strongly on the superomedial angle of the bone and the shoulder girdle was slightly elevated.

The integrity of the serratus anterior muscle, difficult to determine clinically in this instance, was demonstrated by electrical stimulation. All the other scapulohumeral and thoracohumeral muscles were normal. Paralysis of the trapezius was the sole defect.

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Inman, Saunders and Abbott, who investigated the forces and the functions of the muscles about the shoulder joint, state that the trapezius acts as three separate components—upper, middle and lower. The upper trapezius with the levator scapulae and the upper digitations of the serratus anterior support and elevate the shoulder and comprise the upper group of muscles necessary for scapular rotation, without which full abduction of the shoulder is impossible. Rotation is strongly assisted by the lower trapezius and the lower digitations of the serratus anterior. The function of the intermediate trapezius, assisted by the rhomboids, is to draw the scapula toward the midline and fix it there so that it will pivot when acted upon by the trapezius and serratus anterior.

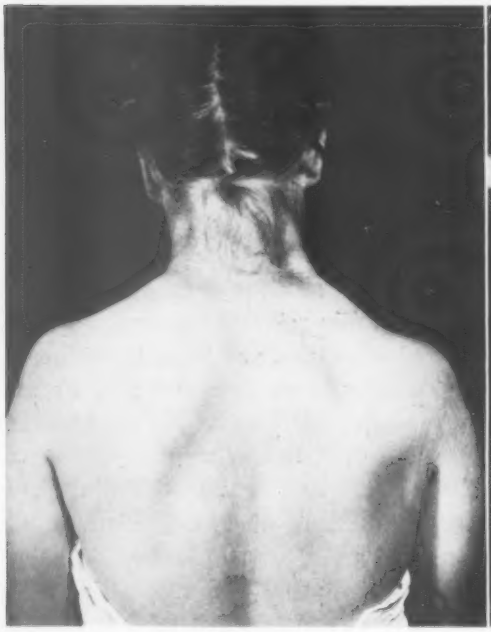

FIG. 1

FIG. 2

FIG. 1.—Note dropping of the right shoulder and the profile of the hypertrophied levator scapulae.
FIG. 2.—The maximum amount of active abduction.

In the case of Mrs. B. the upper trapezius failed in its role of supporter and the result was a drop-shoulder. The constant unopposed contractions of the levator scapulae plus the weight of the arm created a counter rotation of the scapula that was most evident when the patient was carrying a heavy object. In a normal person, when abduction of the arm is conditioned by eliminating scapular elevation and rotation, motion is confined to the scapulohumeral joint and is limited to 80 degrees (Inman and Abbott) In our patient, failure of the intermediate trapezius to fix the scapula jeopardized

the function of the serratus anterior. Abduction was 90 degrees; only slightly more than that possible at the scapulohumeral joint alone. The power to rotate the scapula was almost completely lost, though only the trapezius was paralysed.

Such gross disablement does not always follow division of the spinal accessory nerve. Sacrifice of the nerve is common in block dissection of the neck. Atrophy of the trapezius muscle follows, accompanied by varying degrees of drop-shoulder. None the less, the majority of these persons are able to

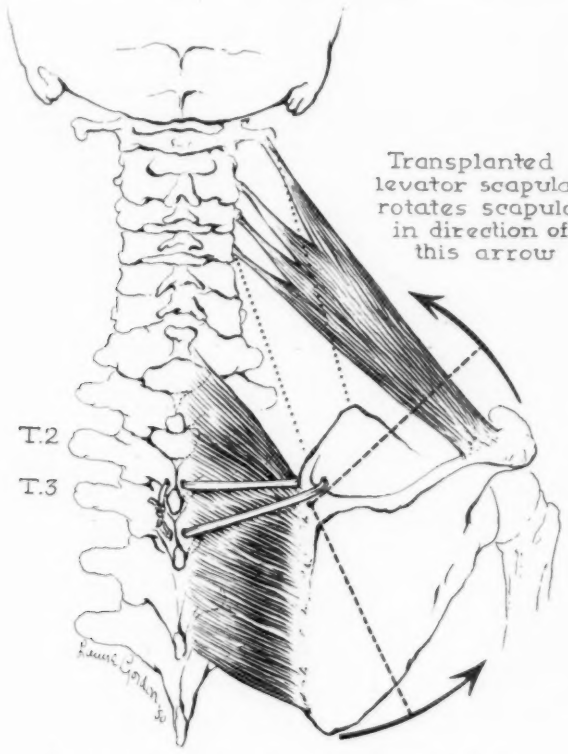

FIG. 3.—Drawing of operation. The scapula is anchored to the spines of the second and third dorsal vertebrae by the fascial sling which passes through the vertebral margin at the root of the spine. The insertion of the levator scapulae is transplanted to the outer end of the spine. The scapula can then be pivoted by the serratus anterior and the transplanted levator scapulae about the point of fixation as indicated by the arrows.

abduct the arm fully although the power of abduction is reduced. Explanation of this is not clear. The trapezius may often derive additional innervation from the cervical plexus. These nerves, independent of the spinal accessory, would prevent complete loss of function of the muscle. However, most

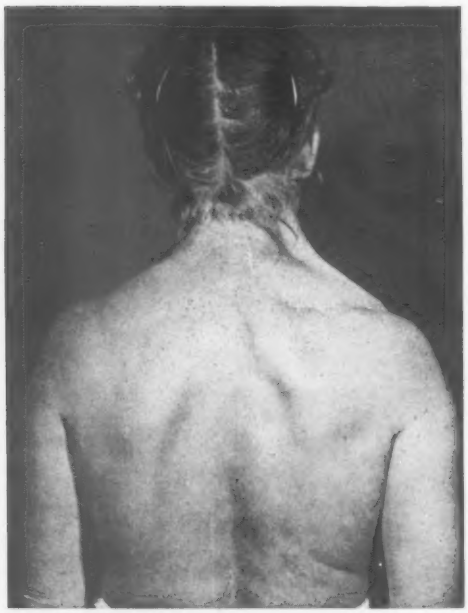

FIG. 4.—Improved resting posture of the shoulder. The transplanted levator scapulae can easily be seen (one year postoperative).

anatomists believe that the cervical branches to the trapezius are proprioceptive and not motor. On the other hand if, in spite of complete trapezius paralysis, the remaining muscles were able to fix the scapula on the chest wall, the serratus anterior could act to produce the desired result. This is the explanation that seems most likely, when the patients are examined. This aspect is still under investigation.

Mrs. B.'s operation was designed to correct the lack of fixation of the scapula on the chest wall and to supplement the forces necessary for rotation (Fig. 3). With the scapula in the normal resting position and counter-rotation corrected, its superomedial angle was tethered to the spines of the second and third dorsal

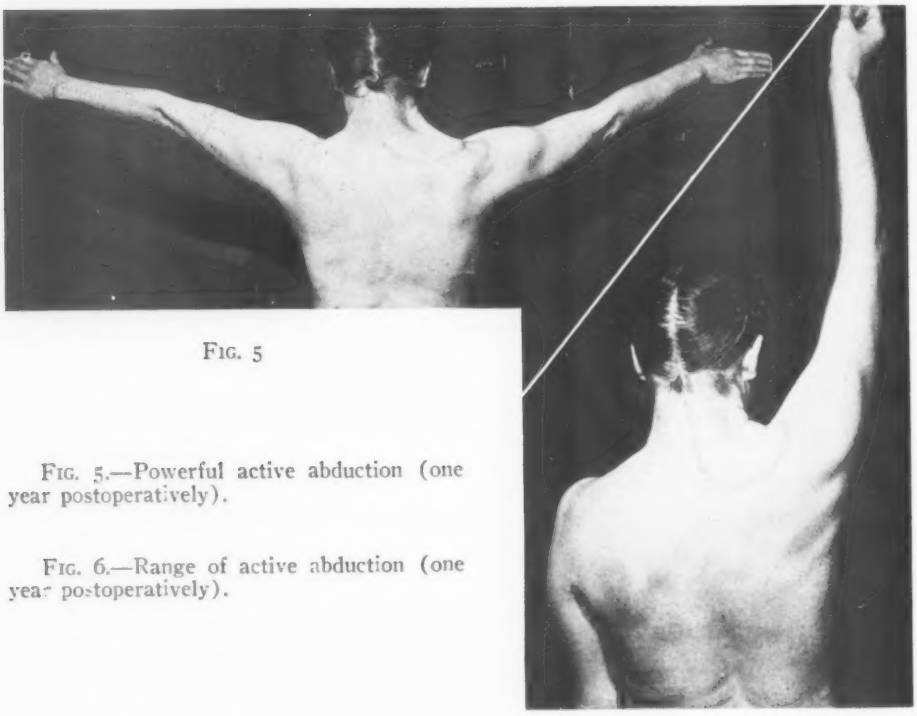

FIG. 5

FIG. 5.—Powerful active abduction (one year postoperatively).

FIG. 6.—Range of active abduction (one year postoperatively).

FIG. 6

vertebrae by a two-inch strip of fascia lata. Directed downward and medially, this opposed the downward rotating pull of the weight of the arm and fixed the scapula in the functional position normally accomplished by the central portion of the trapezius. The distal insertion of the levator scapulae was then detached, displaced laterally, and re-implanted into the spine of the scapula adjacent to the acromion to act as a substitute for the upper trapezius. The abducted arm was encased in a plaster spica for eight weeks, when active exercises were begun. An excellent exposure was obtained through the L-shaped incision whose scar is seen in Figure 4.

The operation was entirely successful in achieving the desired result. With its vertebral margin fixed by the fascial sling, the scapula, when acted upon by the force couple of the serratus anterior and the transplanted levator scapulae, accomplished a type of movement which approached normal in range, strength and direction. The patient returned to the farm and was able once again to assume her full share of responsibilities. Her ability to carry heavy objects in the right hand was restored and she regained an almost complete range of movement in a very powerful shoulder (Figs. 4, 5 and 6).

The complexity of the problem of shoulder dysfunction which results from paralysis of the trapezius and the simplicity of its solution by an operation based on appreciation of the functional anatomy of the joint prompt us to report this case.

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ANNULAR PANCREAS*

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ANNULAR PANCREAS is a sufficiently uncommon and infrequently reported anomaly so that its presence is rarely suspected. This is the more true because it is probably the only congenital anomaly of the gastro-intestinal tract which usually produces symptoms late in life. There have now been reported some 50 instances of this anomaly, most of them purely anatomic descriptions of necropsy findings. The malformation was given its aptly descriptive name in 1862 by Ecker,[10] writing in Henle's Zeitschrift. In the course of an anatomic demonstration on the body of a young man, he found "a strip of glandular substance which lay across the descending portion of the duodenum. Closer investigation showed this to be the anterior portion of a ring derived from the head of the pancreas which surrounded the descending portion of the duodenum and was formed by uninterrupted glandular tissue." This "ring formigen" portion of the pancreas contained one duct which communicated superiorly and posteriorly with the main pancreatic duct. Schirmer[20] in his 1893 dissertation on the pancreas erroneously stated that Tiedemann,[24] Bécourt,[1] and Moyse[19] had observed cases of annular pancreas and reference to these authors is still made in most discussions of annular pancreas. Review of these reports, however, shows that the writers were either discussing the comparative anatomy of the pancreas and the occurrence of annular pancreas among the birds or else were discussing carcinoma of the pancreas or other acquired encircling lesions in this area. There are 14 recorded instances of operation for annular pancreas. Most reports are of one or two cases, individual experience with a lesion of this rarity being necessarily limited. Among the most helpful discussions are those of Howard,[13] McNaught,[18] Gross and Chisholm,[12] Goldyne and Carlson,[11] and Burger and Aldrich.[4]

At operation or autopsy, one finds a thin flat band of grossly recognizable and apparently normal pancreatic tissue anterior to the duodenum in its second portion. This band is continuous with the head of the pancreas on both the convex and the concave surfaces of the duodenum, which is thus encircled by pancreatic tissue. In the instances which we have seen, there is no point of demarcation between this anterior band and the head of the pancreas with which it is continuous. On the other hand, in many of the instances previously described, and in one of our cases, the band of misplaced pancreatic tissue lies loosely upon the duodenum and may be lifted away from it. It is thus quite different from that other anomaly, *aberrant pancreas*,[8] in

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which an isolated nodule of pancreatic tissue lies buried in the wall of the stomach, jejunum, or ileum, frequently submucosally.

The anterior band of pancreatic tissue often contains a fairly large duct which has most often been reported to connect with the main pancreatic duct of Wirsung. In Cunningham's[6] case, the abnormal portion of pancreas had a duct system of its own which entered into the common bile duct on a slightly posterior plane to the entrance of the main pancreatic duct.

The generally accepted embryologic explanation for this malformation depends upon the development of the pancreas from two anlagen, dorsal and ventral, making their appearance as buds from the midgut in the 3 to 4 mm. embryo before rotation of the gut takes place. In the course of development the dorsal anlage usually forms the body and tail of the pancreas and part of the head and uncinate process. The ventral anlage contributes to the head of the pancreas. Its duct joins with the distal portion of the dorsal duct to form the duct of Wirsung. The proximal portion of the dorsal duct becomes the accessory duct of Santorini. It is postulated that, in rotation of the duodenum, pancreatic tissue from the ventral anlage may be carried with the second portion of the duodenum, subsequently fusing its free end superiorly to the head of the pancreas, creating the annular deformity. Chapman and Mossman[5] are inclined to think that the ring is produced by overgrowth of the ventral anlage around both sides of the duodenum, fusing to the head of the pancreas. Weissberg[28] found an annular pancreas in a 16-millimeter embryo (approximately six to seven weeks). At this early stage the annular ring was quite well formed. It appeared to originate from the ventral anlage and had expanded about the duodenum. On the posterior portion of the duodenum, the pancreatic tissue lay beneath the peritoneum and actually in the wall of the bowel. Weissberg feels that neither abnormal persistence of the ventral anlage nor anomalous fixation and rotation with the duodenum are responsible for the formation of annular pancreas, but that there may be simply an unusual growth of one of the ducts of the pancreas itself which the local mechanical situation may direct in this way.

CLINICAL CHARACTERISTICS

In many instances, the condition is asymptomatic, and the annular pancreas is described as a curious, coincidental necropsy finding. In 14 cases—17, with the inclusion of the three herein reported—symptoms were produced of severity sufficient to warrant operation. In one of our patients (J. K.) and in the case of Lehman reported in 1942, the correct diagnosis was made before operation. Sixteen of the 17 patients developed signs of high intestinal obstruction. The seventeenth was operated upon for peritonitis found to be due to acute pancreatitis. Most of the patients were well on in years when symptoms began, and only four were in the neonatal period. Ten of the 17 patients were male. Only one patient was colored. In the older patients the appearance of indigestion, eructation, and vague epigastric pain preceded the onset of frank intestinal obstruction.

The diagnosis may be made in patients with chronic duodenal obstruction who show by roentgen ray an almost complete, smooth and sharp obstruction of the duodenum to the right of the midline. The diagnoses which have been erroneously made in the past are arterio-mesenteric ileus, cholecystitis, primary carcinoma of the duodenum, cicatrizing peptic ulcer, malrotation of the bowel, duodenal atresia, etc.

The barium meal, although rather characteristic, has often failed to lead to the diagnosis of annular pancreas. In one of our cases the roentgenologic report was "unusual dilatation of the distal third of the stomach." In Lehman's case[15] the roentgenographic diagnosis was first "a polyp of the second portion of the duodenum," and this then changed to "constricting ulcer."

In view of the rarity of annular pancreas, surgeons are justified in giving greater consideration to such rare conditions as carcinoma of the duodenum in adults and duodenal atresia in newborn infants. In 20,000 autopsies at the Johns Hopkins Hospital, annular pancreas has not been noted even once.

TREATMENT

One is dealing with a mechanical duodenal obstruction and operative interference is required for relief. Direct attack upon the constricting portion of the ring has been attempted seven times in all, including the present cases. In these instances, the ring has been divided, separated from the duodenum, and a portion resected.

Six of these seven patients survived, the seventh dying on the ninth day after operation. No autopsy was performed, but bile-stained drainage appeared from the wound. Three of the six surviving patients had persistent pancreatic drainage, and one of the three had a second operation for drainage.

In Lehman's case[15] of resection of the ring, the patient survived without fistula formation, but had persistent symptoms and roentgenograms showed persistent duodenal deformity. Similarly, one of our cases, despite the readiness with which the ring was resected, developed progressive duodenal obstruction and had to be operated upon a second time. Two of the seven patients also had a plastic procedure on the duodenum—one, the fatal case mentioned, and the other with successful outcome.

In summary, direct attack upon the annular pancreas has now been employed seven times with one straightforward cure, one death, three instances of prolonged pancreatic drainage, and two instances of persistence of symptoms. In addition, in one case,[3] annular pancreas was associated with acute hemorrhagic pancreatitis for which simple drainage was instituted. The annular pancreas was discovered at autopsy.

The second alternative, a by-pass operation, was historically the first method of treatment[27] and has now been performed ten times. There have been five gastro-enterostomies, one gastric resection in a patient who had a coincident peptic ulcer,[7] three duodenojejunostomies, and one gastro-duodenostomy. Three of the patients with gastro-enterostomy died—two of upper respiratory infections and one several hours after operation, presumably of

shock. All three of these deaths occurred prior to the days of antibiotics and multiple transfusions. One of our patients with a duodenojejunostomy had previously had a partial resection of the pancreatic ring without improvement.

CASE REPORTS

Case 1.—J. K., J. H. H. No. 317888. This was a 67-year-old man admitted to the Johns Hopkins Hospital on January 21, 1949. Fifteen years prior to admission the patient first developed intolerance to fatty food. Three years prior to admission he developed epigastric pain after meals, at first relieved by soda but later becoming more severe and persistent. In recent months he had vomited frequently and had often complained of headaches and a foul taste in his mouth. He had lost approximately 10 pounds in weight.

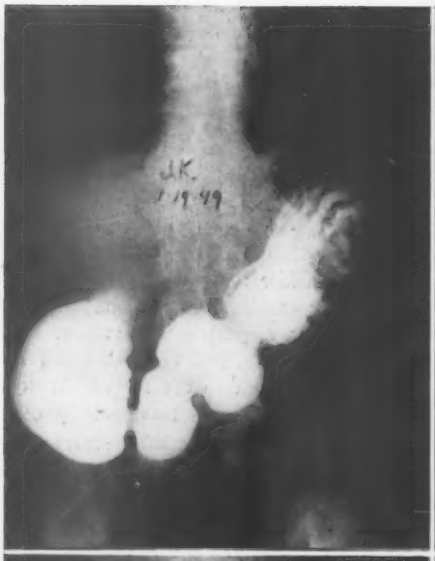
Physical examination revealed an undernourished white male who appeared chronically ill. There was a definite bulge in the right upper quadrant of the abdomen where peristaltic movements could be seen. There was slight epigastric tenderness, but no masses or muscle spasm could be felt. The liver was palpable two fingersbreadth below the right costal margin on inspiration. Blood and urine studies were all within normal range. Gastric analysis showed free hydrochloric acid of 18° and total acidity of 52°, rising to 134° and 172° respectively after histamine. A gastro-intestinal series showed what at first was thought to be almost complete pyloric obstruction with "unusual dilatation of the distal third of the stomach" (Fig. 1, A and B; Fig. 2). A small focus of calcification seen in the right upper quadrant was thought to be a calcified lymph node. In the discussion of the patient by the members of the hospital staff it was pointed out that the "dilated third portion of the stomach" seen on the gastro-intestinal series might well be duodenum. Among the diagnoses suggested were chronic duodenal ulcer, chronic cholecystitis, arterio-mesenteric ileus, and annular pancreas (advanced as a definite diagnosis by one of us).

Operation was performed (A. W., Jr.) on January 27, 1949, through a transverse incision. The first portion of the duodenum was found to be dilated and flabby. The stomach and pylorus appeared normal. The descending portion of the duodenum was mobilized. Just inferior to the point at which the common duct entered the duodenum, a broad band of pancreas completely encircled the duodenum, causing almost complete obstruction. Beyond this band the duodenum appeared normal in size. The annular portion of the pancreas was approximately two centimeters wide and one-half to one centimeter thick. It was elected to remove the anterior and lateral portions of the encircling band of pancreas. This was easily accomplished. No large pancreatic duct was identified. The cut ends of the pancreatic ring were transfixed with mattress sutures of silk. The lumen of the duodenum then appeared open, and it was thought that the obstruction was relieved. A drain was placed in the vicinity of the cut ends of pancreas and brought out through a stab wound in the right flank. Pathologic examination of the resected portion of the pancreatic ring revealed only normal pancreatic tissue.

The first week of the postoperative course was uneventful. Very little drainage was noted. On the seventh postoperative day the patient became distended and vomited, and on successive days he vomited several times. On the ninth day after operation, overnight gastric retention was found to be 400 cc., and he had a serum amylase of 518 mg. per 100 cc. A Levine tube was passed into his stomach and attached to constant suction. He was maintained on intravenous feedings. On the eleventh day the serum amylase was 756 mg. per 100 cc. The Levine tube was removed on the sixteenth day and he was begun again on a soft diet by mouth. The serum amylase thereafter was at normal levels. He was discharged on the nineteenth postoperative day, at which time he was without complaint and taking a full soft diet well.

Shortly after his return home he began again to have symptoms of epigastric fullness and he vomited on several occasions. On one visit to the hospital, one liter of

FIG. 1-A



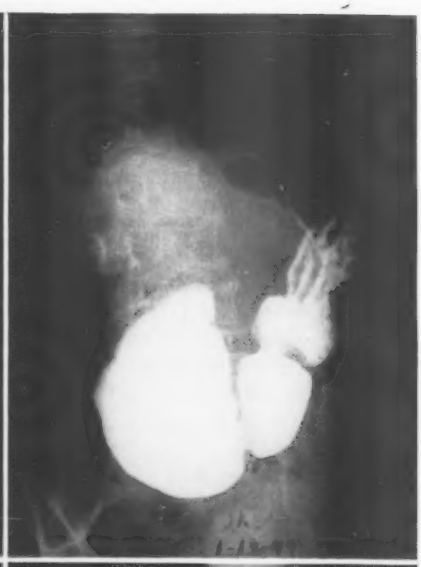
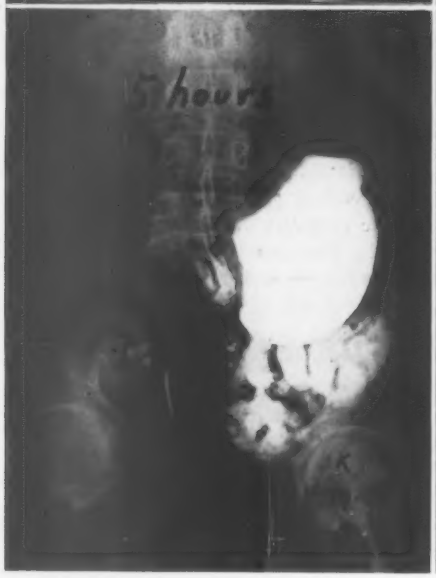


FIG. 1-B

FIG. 2



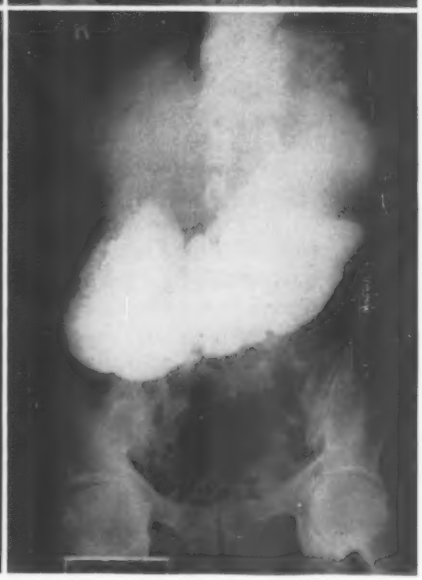


FIG. 3

FIG. 1.—(Case I) J. K., white male, age 67, No. 317888 (A and B) Anteroposterior and lateral views of the barium swallow. There is a tremendous dilatation of the duodenum ending quite abruptly at a smooth obliquely transverse obstruction in the second portion. There is no puckering or filling defect.

FIG. 2.—(Case 1) Five hour film demonstrates degree of gastric retention.

FIG. 3.—(Case 1) Barium swallow 25 days after resection of pancreatic ring—duodenum and stomach are more dilated than before.

gastric contents was removed by lavage. A gastro-intestinal series was obtained and showed the original dilatation of the duodenum plus marked additional distention of the stomach which had not been present before (Fig. 3. He was re-admitted to the hospital on March 5, 1949, and the original incision was reopened (A. W., Jr.). The duodenum was again seen to be markedly dilated in its first portion and had not changed in size. The pylorus, however, had become patulous and would admit three finger tips easily. The stomach had increased greatly in volume, and the gastric wall appeared hypertrophied. A thick band of scar tissue was found in the region of the excised annular portion of the pancreas. This thick band of scar passed anterior to the duodenum and ran from the transected end of the pancreatic ring to the lateral abdominal wall at the site of the stab wound made for the drain. The duodenum was still narrow at the site of the original obstruction, but the present obstruction seemed caused by the dense band of scar tissue. The jejunum was identified at the ligament of Treitz, and a loop approximately 40 cm. distal was brought up anterior to the colon and anastomosed to the anterior surface of the dilated first portion of the duodenum with two rows of interrupted sutures of fine silk.

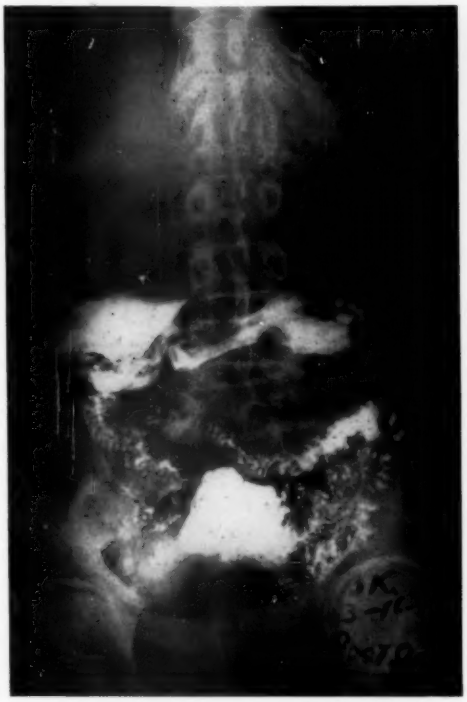


FIG. 4.—(Case 1) Barium swallow (one hour film) 11 days after second operation—duodeno-jejunostomy. The patient has remained well since.

Postoperatively, the patient was maintained for three days on intravenous feedings while constant gastric suction was applied to an indwelling Levine tube. Following this period he was fed a gradually increasing liquid diet until the tenth postoperative day when he was placed on a full soft diet. He was discharged from the hospital, asymptomatic, on March 18, the fourteenth postoperative day.

On one occasion in the Out-Patient Department, he complained of epigastric fullness but a gastric aspiration at that time revealed only 100 cc. of fluid in his stomach. A gastro-intestinal series (Fig. 4) showed a satisfactorily functioning duodeno-jejunostomy, though the stomach was still dilated. He was last seen in June, three and one-half months after the second operation, when he felt entirely well and had gained 17 pounds since his discharge from the hospital.

Comments. This was a 67-year-old white male who had a 15-year history of indigestion and a three-year history of more specific symptoms. Annular pancreas was diagnosed from the roentgenograms. Division of the annulus and resection of a portion of it produced a local acute pancreatitis resulting in a dense scar obstructing the duodenum more completely than before. Duodeno-jejunostomy afforded complete relief.

Case 2.—M. G., No. A-68776. The patient was a full-term white female whose delivery was uneventful. Beginning shortly after birth she vomited thin, bile-stained material

at frequent intervals and was able to retain nothing by mouth. Physical examination revealed an alert white female infant, externally normal, and presenting no other evidence of any congenital malformation. Weight on transfer to the surgical service, when she was three days old, was 2770 Gm. Roentgen ray examination (Fig. 5) showed a large, dilated stomach, no dilated loops of intestines, and a moderate amount of gas in the large bowel. The preoperative diagnosis was partial high intestinal obstruction secondary

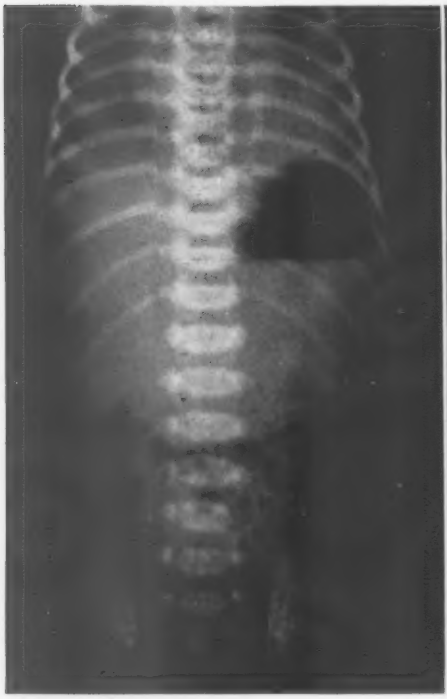


FIG. 5

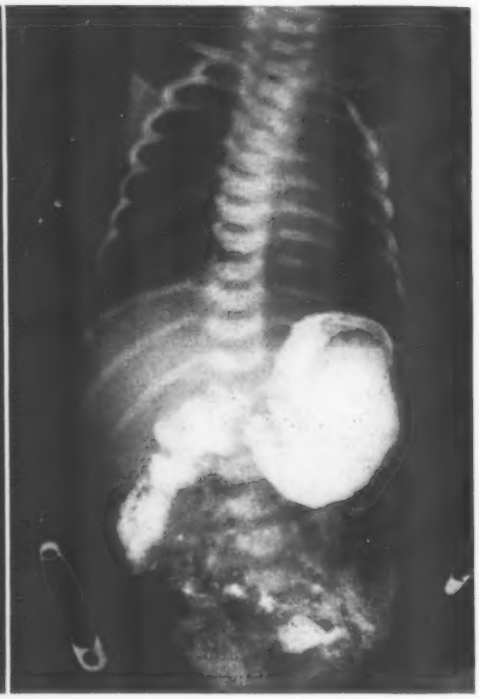


FIG. 6

FIG. 5.—(Case 2) M. G., white female, age three days, No. A-6876. Roentgenogram on third day of life showing stomach dilated with air. The presence of some air in the lower intestinal tract demonstrates the obstruction to be incomplete.

FIG. 6.—(Case 2) Roentgenogram of barium swallow taken 14 days after duodeno-jejunostomy. Barium passes at once from the still dilated duodenum into the jejunum whose plicae circulares are plainly seen.

to a partial duodenal diaphragm, malrotation, or some other congenital malformation.

On February 17, 1949, under open-drop ether anesthesia, laparotomy was performed (M. M. R.) through a transverse incision in the right upper quadrant. When the abdomen was opened the small intestine was found to be collapsed almost completely, but it was not quite as small as in a true atresia. There was meconium in the colon. No abnormality was found in the jejunum. The stomach, which had been emptied by suction, was large and thick walled, and the proximal duodenum for 2 to 3 cm. was almost as large as the pre-pyloric region of the stomach (Fig. 5). Distal to this dilated portion of duodenum, the duodenum was encircled by a band of pancreas about 1 cm. wide, beyond which the duodenum was no larger than was the jejunum at the ligament of Treitz—in other words, smaller than normal. A brief attempt was made to dissect the pancreas from the duodenum, but it was so intimately attached that the operator

thought the dissection would be fraught with danger. Accordingly, a duodeno-jejunostomy was performed, bringing up anterior to the transverse colon a loop of jejunum some distance below the ligament of Treitz. A two-layer anastomosis was accomplished, using an outer continuous suture of 5-0 silk and an inner continuous suture of 4-0 catgut. The patient's condition throughout the procedure was good, and she received 100 cc. of whole blood during the course of the operation. The wound was closed with silk without drainage.

The patient's postoperative course was uneventful except for the development of edema of the epiglottis secondary to the indwelling Levine tube which was used for gastric suction for 48 hours. This difficulty vanished with the removal of the tube. The incision healed per primam. The patient began to take oral feedings well on the fourth postoperative day and was discharged from the hospital on March 8, 1949, 19 days after operation, apparently well and gaining weight satisfactorily on a standard formula. A G. I. series performed prior to her discharge (Fig. 6) showed good function of the anastomosis, though the duodenum was still dilated.

Comments. This was a thre-day-old female with incomplete duodenal obstruction due to an annular pancreas. Duodenojejunostomy gave complete relief.

Case 3.—R. L., No. A-52869. The patient was born in a doctor's office in a rural district of Maryland, in an otherwise normal delivery. The child and the mother were apparently sent home the same day. On the third day the doctor was told that the baby was vomiting and had passed no stools. The infant was found to be in critical condition, jaundiced, dehydrated, feeble, and vomiting small amounts of greenish mucus. Temperature was 102°. The baby was given a subcutaneous infusion and transferred to the Harriet Lane Home. He was then six days old. His birth weight had been 6 lbs. 9 oz. Vomiting was said usually to occur three hours after each feeding. One sibling, a girl of four, had previously been diagnosed as a Mongolian idiot. Physical examination showed the baby to a a scrawny-looking undernourished infant weighing only 1950 Gm. He was quiet, obviously ill, and deeply jaundiced. A plain roentgenogram of the abdomen showed a large collection of air in the stomach with no gas visible in either the small or large intestines (Fig. 7). Barium administered by mouth remained within the stomach for four or five hours (Fig. 8). Surgical consultation was requested at this point, and the general consensus was that the baby had a congenital duodenal obstruction which required operative relief. Exploration was performed (M. M. R.) on March 23, 1947, when the patient was 8 days old. The abdomen was entered through a transverse incision just above the umbilicus. The stomach was much enlarged and thick-walled. All the intestines were delivered, and the small bowel followed up from the cecum, which was peculiarly situated near the midline and rather high up. There was no fixation of the mesentery of the small bowel, the bowel being suspended largely by its vessels. There was no ligament of Treitz. The duodenum coiled and kinked several times on itself in the right upper quadrant, the coils and kinks being held together by what could only be described as organized adhesions. Some of these were opaque and suggested a reaction of inflammatory origin more than anything else. All of the small bowel was tiny and contained no air or meconium, while the proximal duodenum was as large as the pyloric antrum of the stomach. The distal duodenum was clamped off with a rubber shod Allis clamp about two inches below the stomach, and the segment proximal to the clamp injected with saline solution under pressure. The duodenum was distended in this manner to the size of one's little finger, until it was extremely tense. The fluid, however, did not run into the stomach, and by inverting the pyloric antrum with one's finger one could palpate the proximal end of the tensely distended saline-filled segment of the duodenum. The level at which this duodenum ended blindly was marked anteriorly by the passage over it of the portal vein. This was unmistakable and readily demonstrated. The superior and inferior mesenteric vessels joined and the

portal vein formed by their junction passed anterior to the duodenum precisely at the level of what must have been an intrinsic obstruction. At this point, too, the pancreas passed around the duodenum as an annular pancreas, the anteduodenal portion of which was just at the level of the abnormally situated portal vein. The annular portion of the pancreas connected uninterruptedly with the head of the pancreas above and below (Fig. 10A). It seemed impossible for any extrinsic obstruction to have resisted the great hydrostatic pressure induced by the injection of saline into the isolated duodenal loop below the obstruction. The loop of duodenum which was injected did not contain any bile. We were forced to the conclusion that this was a complete atresia. This feeling was bolstered by the report from the Pathological Department that the rectal discharge before operation, which was in the nature of a greenish mucous plug, did not contain any squamous epithelium or lanugal hair. The gallbladder was normal and distended with very dark bile. No attempt was made to follow up the common duct, although it appeared to enter the duodenum just proximal to the obstruction. The duodenum proximal to the obstruction was as dilated as the stomach, and could hardly be told from it. The stomach was anastomosed to the duodenum just beyond the obstruction, using

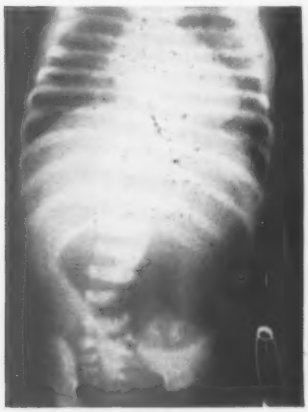


FIG. 7

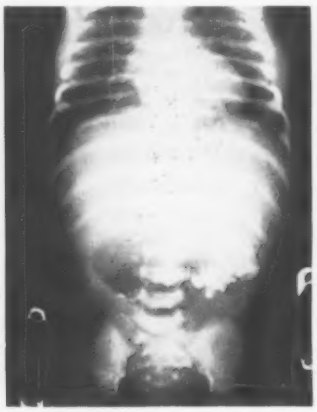


FIG. 8

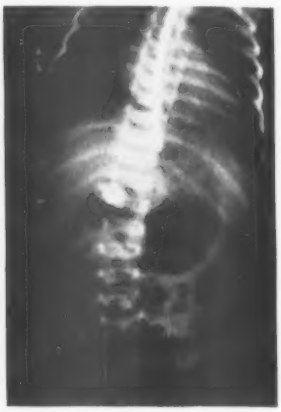


FIG. 9

FIG. 7.—(Case 3) R. L., white male, age eight days, No. A-53869. Preoperative roentgenogram showing tremendous dilatation of stomach and duodenum and absence of air elsewhere in the intestine.

FIG. 8.—(Case 3) Barium has unwisely been administered. The picture is less clear than before. The barium swallow is an unnecessary and dangerous measure in the diagnosis of neonatal intestinal obstruction.

FIG. 9.—(Case 3) Roentgenogram two weeks after gastroduodenostomy. The intestinal tract contains air throughout. A little barium persists in the still dilated duodenum. (The child is well two and one-half years after operation.)

a double row of sutures—outer of continuous ooooo silk and inner of continuous ooooo catgut.

The colon was found now to present anomalies both of rotation and of fixation. Traced upward from the pelvis the colon went straight up the abdomen on the right side, crossed over to the left, made a rather large loop and passed behind itself to emerge on the right side with little more than the cecum to the right of the vertically ascending limb first described. The colon was fixed in this position by fibrous bands which again suggested adhesions like those which held the duodenum. As these bands were released and the colon uncoiled, its mesentery was found to have no fixation at all.

Postoperatively the child was maintained on parenteral fluids. Nothing was administered by mouth, and constant gastric suction was employed for three days until serial

roentgenograms showed air passing on into the bowel beyond the level of the anastomosis (Fig. 9). This occurred on the third day; glucose was then started by mouth and the child gradually given increasing feedings. He was discharged on April 26, 34 days after operation, taking a full diet and weighing 2620 Gm. as compared to his admission weight of 1955 Gm. He has grown well in the two and a half years since operation and is apparently normal in all respects.

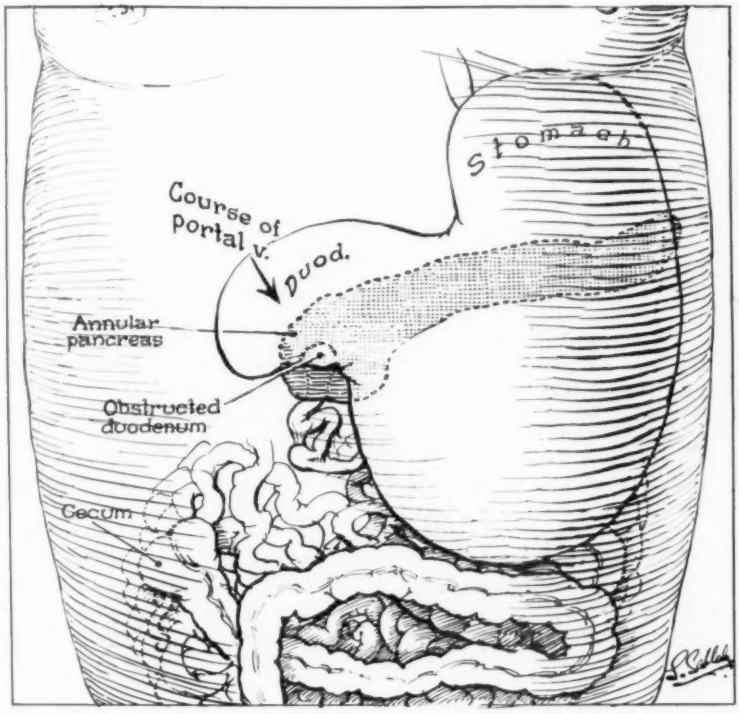


FIG. 10.—(Case 3) R. L., white male, age eight days, No. A-53869. Operative findings: The stomach and first portion of the duodenum are tremendously dilated. There is an annular pancreas surrounding the duodenum at a level coinciding with a complete intrinsic duodenal obstruction. (The portal vein passed anterior to the duodenum just proximal to the annular pancreas, but the artist has omitted this for simplicity.) The duodenum does not pass over to the left behind the superior mesenteric artery and is repeatedly kinked on itself by adhesions. There is a malrotation of the colon. After the peritoneal bands which held it in this position were released the colon was found to swing freely from a long mesentery attached only at its root.

Comments. This was an eight-day-old infant in whom deep jaundice complicated the picture of complete duodenal obstruction. Jaundice, presumably due to back pressure, is not rare in such situations. An annular pancreas was associated with malrotation of the intestine, complete duodenal atresia, and passage of the portal vein anterior to the duodenum. Gastro-duodenostomy relieved the obstruction, and division of mesenteric and peritoneal bands corrected the malrotation.

DISCUSSION

These three cases provide fairly characteristic instances of annular pancreas. The two infants were typical of patients with neonatal duodenal obstruction, whatever the cause. One infant presented a combination of malformations—namely, an annular pancreas and atresia of the duodenum—previously described by Vidal[27] in 1905 in an operation on a newborn in whom gastroenterostomy was successful. In addition, our patient had a malrotation of the colon, which has been described by Gross and Chisholm[12] in connection with annular pancreas. The portal vein in our patient crossed anterior to the duodenum. In our two infants, a flat diagnosis of annular pancreas excluding all other possibilities would have been untenable.

The adult was typical of older patients previously reported with annular pancreas. In this instance, the diagnosis was suggested before operation and could readily be supported on the basis of the history and roentgenologic findings. One aspect of the history of the condition in adults remains unexplained—the late onset of symptoms and the slow progression of symptoms, once present.

In all three of these cases, a by-pass operation was the treatment of choice. A gastro-duodenostomy was performed in the earliest case because of the malrotation of the viscera and because the entire duodenum lay on the right side of the abdomen, readily adjacent to the stomach. In the other two cases, a duodenojejunostomy was performed because of the dilatation of the duodenum proximal to the annular pancreas which rendered such an anastomosis technically easy. In neither of the infants would resection of the pancreatic ring have been a feasible procedure, in one because of dense adherence between the duodenum and the pancreas, in the other because of the associated-atresia of the duodenum. In the adult patient, resection of the ring was performed first but without relief of the obstruction. The presence of scar tissue exactly at the site of the previously resected ring gives credence to the suggestion that leakage of pancreatic secretions stimulated the dense scar formation. Prompt and immediate relief from obstruction was obtained in all three patients with the formation of short circuiting anastomoses.

SUMMARY

Three cases of annular pancreas are reported, two infants and an adult. Operation was performed in each instance with success. Division of the pancreatic annulus introduces the hazards of pancreatic fistula, or of pancreatitis. Duodenojejunostomy is probably the procedure of choice.

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NEW ABSORBABLE HEMOSTATIC BONE WAX

EXPERIMENTAL AND CLINICAL STUDIES*

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IN RECENT YEARS a number of new absorbable hemostatic materials have been developed. These were studied primarily for use in neurosurgery but have now been widely employed in general surgery as well as genito-urinary, orthopedic and gynecologic surgery and even in obstetrics. One of us (V. K. F.) had the opportunity to work, under OSRD contract, on oxidized cellulose, *i.e.*, absorbable gauze and cotton. The specific styptic action of these agents, apart from their hemostatic property has been obvious in clinical use.

It was suggested by Henderson,[6] therefore, that advantage might be taken of this specific hemostatic action in conjunction with another kind of hemostatic packing also studied primarily by neurosurgeons, *i.e.* bone wax. Horsley's note in the British Medical Journal in 1892[8] described the formula of bone wax as seven parts beeswax, one part almond oils, and salicylic acid 1 per cent. This had the desired physical properties, was kept in stoppered bottles and sterilized by boiling. The use of this material for control of bleeding from bone became a standard neurosurgical technic and was also used when indicated in other fields of surgery, such as chest surgery, for example, when the sternum is split longitudinally.

Ordinary bone wax is effective by virtue of its tamponade action, but it has no inherent hemostatic quality. In addition, it is not soluble in the body fluids and thus remains at the site of implantation for long periods of time. That portion of it which eventually is removed is probably carried away through the action of phagocytic cells. As a result, the wax acts as a foreign body, tending to promote infection and in itself causing a low grade inflammatory reaction in the tissues about the site of implantation.

Oxidized cellulose and the new water soluble ointment bases offered the possibility of developing a substance which had the same physical properties as the beeswax mixture of Horsley and also was specifically hemostatic and absorbable. At Henderson's suggestion,[6] some of the polyethylene glycols of high molecular weight were chosen as the base. A hemostatic absorbable substance has now been produced and is the subject of this study.

* The work described in this paper was carried out under a grant from the Johnson and Johnson Research Foundation. The experimental materials used in the study were supplied by the Department of Clinical Research, Ethicon Suture Division, Johnson and Johnson, New Brunswick, New Jersey. Submitted for publication December, 1949.

COMPOSITION AND PROPERTIES OF THE NEW ABSORBABLE, HEMOSTATIC BONE WAX

The material is dispensed already sterilized by autoclave, wrapped in scored tinfoil and ready for use in sealed glass tubes of the type familiar to all who have used catgut sutures. It has a consistency somewhat firmer than petrolatum but is definitely less firm and more malleable than ordinary bone wax. Since it melts near body temperature, handling the material for any length of time causes it to become softer. Prolonged handling will eventually cause it to liquify. The material has a uniform, dark brown color.

Because of its greater malleability and softer consistency, this material is more easily applied to the bleeding crevices and openings in bone than the ordinary type of wax. In addition, it does not have the tendency to crumble into small particles, and after it has been applied adheres much more tenaciously to the bony structures. It adheres to rubber gloves somewhat more than ordinary bone wax but this is easily remedied because it is water soluble and can be rinsed off.

It is undoubtedly through the mechanism of tamponade that the new material affects immediate hemostasis, but very soon the known specific hemostatic property of oxidized cellulose comes into effect and a plug composed partly of blood, partly of hemostatic bone wax is formed which prevents further bleeding. The exact chemical mechanism bringing about the hemostatic effect of oxidized cellulose has never been completely worked out. In 1944 Frantz, Clarke and Lattes[3] observed that when oxidized gauze is placed in a wound it soon becomes black, swells and forms a sticky, gelatinous mass and bleeding stops quickly. Whatever the nature of this mechanism, numerous experimental and clinical observations on the properties of oxidized cellulose have confirmed this early finding.

After testing a number of samples of the new bone wax containing different amounts of powdered oxidized cellulose and various combinations of polyethylene glycols, the material described in this paper was finally selected because it was thought to have the best physical and hemostatic properties. The formula of the substance is as follows: Carbowax 1540 — 60 per cent; polyethylene glycol 300 — 15 per cent; oxidized cellulose — 25 per cent. However, it is anticipated that further modifications may be made.

CHEMISTRY, TOXICITY AND CLINICAL APPLICATION

There is no need to recapitulate the laboratory and clinical investigations which resulted in establishing oxidized cellulose as a non-toxic, non-irritating absorbable material.[3, 4] The polyethylene glycols are compounds with the general formula: CH_2 —CH_2OH. The lower members of the series are

$$\begin{matrix} CH_2 \\ | \\ O \\ | \\ CH_2 \\ n \end{matrix} \quad \begin{matrix} \\ \\ \\ \\ —CH_2OH \end{matrix}$$

known simply as polyethylene glycols. Those substances with molecular weights of 1000 or higher are solids and are known as carbowax compounds. All these materials are in reality mixtures of a number of closely related polyethylene glycols and are designated by a number which represents the average molecular weight of the various compounds present. Since the only reactive portions of the molecule are the two hydroxyl groups, 83 per cent to 99 per cent of the molecule, depending upon the molecular weight, is chemically inert. Consequently these substances are remarkably stable and enter into few chemical reactions. They are highly soluble in water. An extensive series of experiments to determine their toxicity was undertaken by Smythe and his collaborators in 1941.[17, 18] These workers have shown that in contrast to the first two members of the group, diethylene and triethylene glycol, which have a low molecular weight and are liquids, the toxicity of the higher members is extremely low. A considerable amount of data on the toxicology of these

TABLE I.—*Absorbable Bone Wax in Rats.*

Number of Animals	Length of Time in Tissues	Gross Appearance		
		Residual Material	Tissue Fluid at Implantation Site	Blood Vessel Engorgement
3	1 hour	Partly liquefied	None	Slight
4	2 hours	Liquid remnant or no residual	Slight or none	Slight or none
3	3 hours	None	Slight or none	Slight
3	4 hours	None	Slight or none	Moderate
9	24 hours	None	No fluid in four; 1 — 3 cc. in five	Slight to none in four; moderate in five with fluid
4	4 days	None	2 cc. fluid in one; no fluid in three	Slight to moderate
9	7 days	None	None	None to moderate
4	10 days	None	None	Slight to none

compounds was accumulated in experiments which included oral, intraperitoneal and topical administration to a number of different kinds of laboratory animals over varying periods of time up to two years. Application to the skin of human beings showed that these substances are no more irritating than a number of other commonly employed bases, such as cocoa butter, glycerol and lanolin. These investigations led to the clinical use of the compounds for topical application. Experimental and clinical confirmatory studies were made by Dodd, Hartmann and Ward[2]; Friedman[5]; Shaffer and Critchfield[15]; Shaffer, Critchfield and Carpenter[16]; Reid and Altemeier[13]; Cochran[1]; Meleney, Johnson, Pulaski and Colonna[11]; Hopkins[7]; and Maynard.[10]

EXPERIMENTAL OBSERVATIONS

Our experimental studies of the new absorbable hemostatic bone wax were undertaken in two phases. In one group of experiments we studied the rate of absorption and irritating effects of the material by means of the standard Lattes-Frantz rat test[9] which has been used extensively in this laboratory in the testing of tissue reactions to foreign materials. Concurrently, experiments

were initiated to determine the effect of the new material on the healing of experimental rib fractures in dogs, comparing these results with the healing observed when ordinary bone wax or oxidized cellulose alone was implanted at the fracture site.

1. Rat Tests. The results of these experiments are summarized in Tables I and II. On gross examination the new bone wax was completely absorbed in all animals at the end of three hours. Microscopic examination at this time, however, revealed particles of oxidized cellulose. At 24 hours some tissue fluid was present at the implantation site in five of the nine animals, but at four days fluid was present in only one out of four. Moderate congestion of the neighboring blood vessels was seen in half the animals (Figs. 1 and 2).

TABLE II.—*Ordinary Bone Wax in Rats.*

Number of Animals	Length of Time in Tissues	Gross Appearance		
		Residual Material	Tissue Fluid at Implantation Site	Blood Vessel Engorgement
1	4 hours	No change	None	None
1	24 hours	No change	None	Slight
4	7 days	Three with cysts averaging approximately 1.5 cm.; one with mass of wax measuring 1.0 cm. in diameter.		Severe

Histologic examination during the first four hours revealed the persistence of oxidized cellulose particles and a moderate number of inflammatory cells. One interesting and unexplained observation was the predominance of eosinophilic leukocytes among these cells. The remainder of the inflammatory infiltrate was made up of polymorphonuclear leukocytes and mononuclear phagocytes. The reason for this early eosinophilic infiltration is not clear because the distribution of leukocytes in the peripheral blood of the rat resembles that of man quite closely.[14] This phenomenon was not observed at 24 hours or later.

The maximum inflammatory reaction was observed at 24 hours, when a moderately intense infiltration of cells was present. About half the cells were polymorphonuclear leukocytes and the remainder were lymphocytes, plasma cells and mononuclear phagocytes. Numerous irregular, basophilic masses were observed which were thought to represent oxidized cellulose particles which had partly dissolved and coalesced. At four days only young granulation tissue with many capillaries was seen, and at seven and ten days denser, more mature fibrous tissues was observed (Figs. 3 and 4).

In the series of control animals in which ordinary bone wax was used, no significant change in the implant was noted in the first 24 hours. At seven days all showed an intense inflammatory reaction, three having cysts containing sterile fluid and numerous particles of wax and a fourth presenting a single, large encapsulated mass of wax. Histologic examination revealed numerous polymorphonuclear leukocytes and an occasional foreign body giant cell (Figs. 5 and 6).

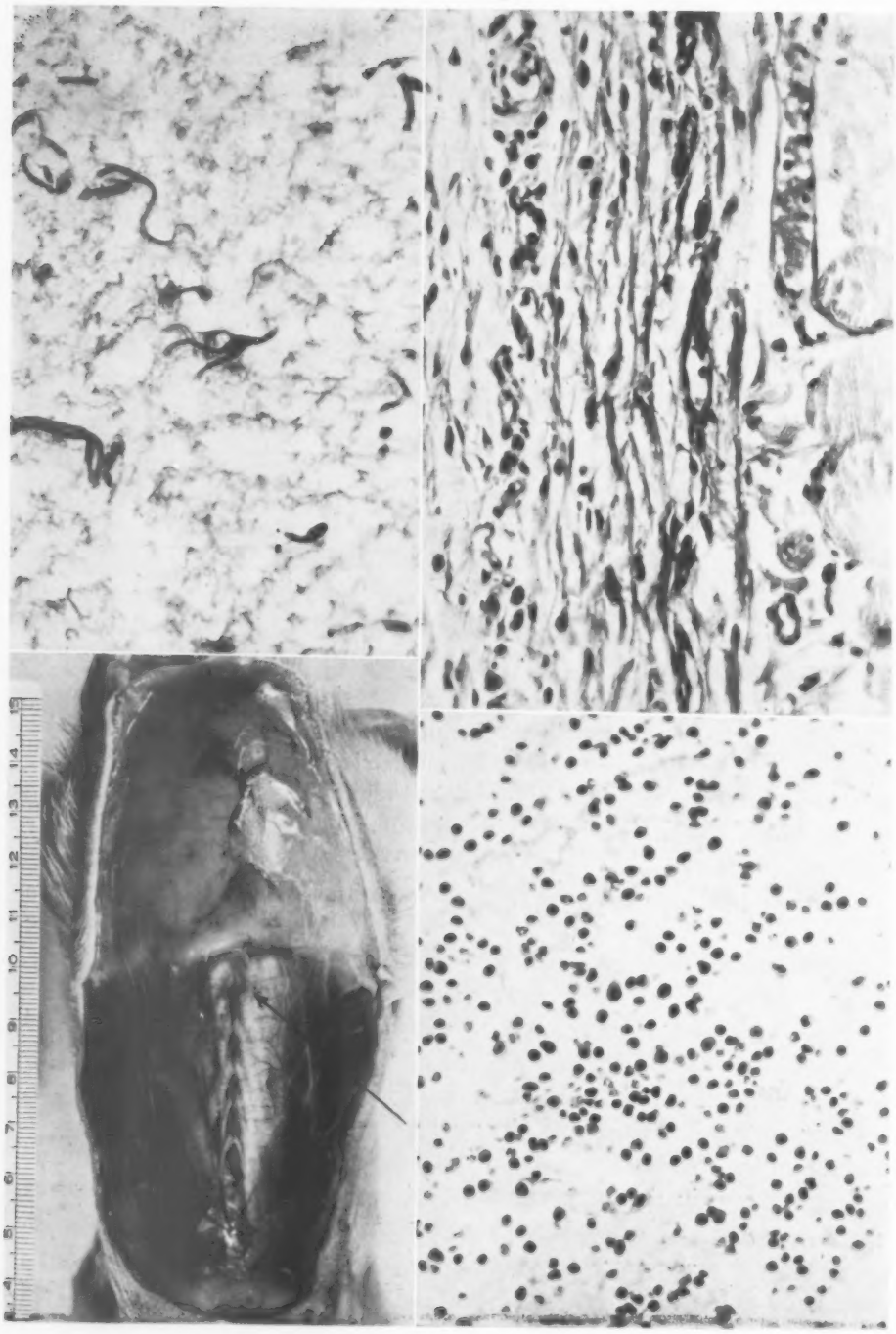

FIG. 1

FIG. 2

FIG. 3

FIG. 4

See legends on opposite page.

2. Rib Implants. The results of the rib experiments in dogs are summarized in Table III. The sixth, seventh, eighth and ninth ribs were used in the first series of animals with implants in order as follows: sixth rib—new bone wax; seventh rib—ordinary bone wax; eighth rib—oxidized cellulose; ninth rib—control. In the second series the sixth, eighth and tenth ribs were employed as follows: sixth rib—new bone wax; eighth rib—ordinary bone wax; tenth rib—control.

TABLE III.—*Experimental Rib Fractures in Dogs*

		X-Ray Evidence of Repair				Microscopic Evidence of Bo..e Formation			
Animal	Number of Days	New BoneWax	Ordinary BoneWax	Oxidized Gauze	Control	New BoneWax	Ordinary BoneWax	Oxidized Gauze	Control
A	2	0	0	..	0	0	0	..	0
B	9	0	0	0	0	+	+	+	+
C	14	0	0	..	0	2+	2+	..	2+
D	23	2+	3+	+	3+	3+	3+	3+	3+
E	30	+	+	..	+	2+	+	..	2+
F	59	union	union	union	union	union	union	union	union
G	91	3+	3+	2+	union	3+	3+	2+	union
H	120	union	union	union	union	union	union	union	union

At the time of necropsy, all wounds appeared to be healing well or had already completely healed. In two cases, autopsied at 59 and 91 days after operation respectively, a number of small particles of ordinary bone wax were scattered widely in the lower portion of the wounds. The pleural surfaces were in all cases intact and appeared pink and glistening. There were no pleural adhesions. The sites of the fractures appeared as smooth, fusiform swellings in the course of the ribs. The degree of immobility already achieved at each fracture site was carefully estimated and recorded. The portion of chest wall containing the fractures was then removed in one segment and immediately placed in a deep-freezing unit. Roentgenograms were taken of all specimens after they had been frozen. The ribs were then sectioned on a small, motor-driven jigsaw and decalcified during a period of from seven to ten days by daily changes of a freshly made up solution consisting of equal parts of 50 per cent formic acid and 20 per cent sodium citrate. Complete and satisfactory decalcification was accomplished by this method with fairly good preservation of cellular details.

FIG. 1.—Absorbable bone wax 24 hours in subcutaneous tissues of back of rat. Complete absorption of material and moderate congestion of blood vessels. (Arrow indicates site of implant.)

FIG. 2.—Absorbable bone wax three hours in back of rat. High power photomicrograph shows fibers of oxidized cellulose and a few inflammatory cells on a background of fibrin strands.

FIG. 3.—Absorbable bone wax 24 hours in subcutaneous tissues of back of rat. Moderately dense infiltration of inflammatory cells consisting of polymorphonuclear leukocytes, plasma cells, lymphocytes and large, mononuclear phagocytes. No residual material.

FIG. 4.—Absorbable bone wax seven days in subcutaneous tissues of back of rat. High power photomicrograph shows cellular connective tissue containing several capillaries.

Active proliferation of new bone from the cambium layer of the periosteum and from the endosteum was first observed in the nine-day fractures. This was noted at all fracture sites at this time. The first definite evidence of calcium deposition, as determined by roentgen rays, occurred at 23 days and bony union at all fracture sites first occurred at 59 days.

Although some difference could be detected histologically in the rate of healing at the different fracture sites, these were not nearly as striking as the

FIG. 5

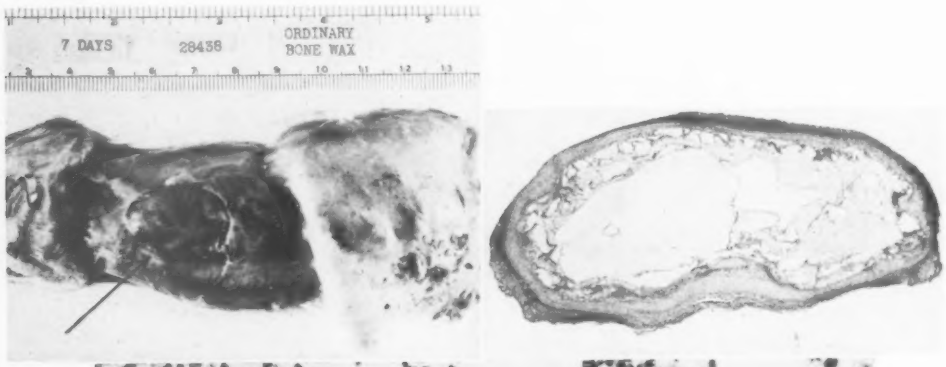

FIG. 6

FIG. 5.—Ordinary bone wax seven days in subcutaneous tissues of back of rat. Upper photograph shows 1.0 x 1.8 x 2.5 cm. cyst (indicated by arrow) which contained thin, brown fluid and particles of wax. Culture negative. Below is low power photomicrograph of cross-section of cyst. See Figure 6 for high power photomicrograph of wall of cyst.

FIG. 6.—Ordinary bone wax seven days in subcutaneous tissues of back of rat. High power photomicrograph of section of cyst shown in Figure 5. Numerous polymorphonuclear leukocytes and irregular, clear spaces which were formerly occupied by particles of wax.

differences in the degree of calcification as demonstrated by the roentgen rays. In some instances there was little or no difference in the histologic appearance of two fractures but a marked and striking difference would be noted in the roentgenogram. This suggested to us that the observed differences were not due so much to differences in the proliferation of osteoid tissue but were rather due to differences in the rate of calcification. In general the roentgen rays showed that the amount of calcified callus present at the site where the new absorbable bone wax was used was either about the same or somewhat less than the amount at the control fracture. Where ordinary bone wax was used the amount of calcified callus was about the same or occasionally somewhat greater than in the control fracture, even though union of the fracture had not yet occurred because of interposition of particles of wax. It is evident from the results that delay in healing occurs with both types of wax but the mechanism appears to be different in the two cases. With ordinary bone wax the delay is probably a result of mechanical factors. The particles of wax remain between the fragments and thus prevent union. With absorbable wax, however, the delay is probably due to alterations in the pH of the tissues. The acidity of the oxidized cellulose undoubtedly lowers the pH of the tissues around the fracture site and thus delays the "alkaline tide" which is essential for the activity of alkaline phosphatase and the deposition of calcium.[12]

On microscopic examination, we observed some irregular basophilic masses in the two-day fracture where the new absorbable bone wax was used. These masses were probably fragments of residual oxidized cellulose. We did not see this material in any of the later fractures. The inflammatory reaction was mild and had completely disappeared after the second day. The ordinary bone wax produced a very characteristic histologic appearance and foreign body reaction, which was easily recognizable as late as the fifty-ninth day. Wherever the wax remained in the tissues, many irregular, clear spaces were present and in these areas would be found numerous, irrgularly-shaped, multinucleated giant cells; lymphocytes; plasma cells and mononuclear phagocytes.

CLINICAL OBSERVATIONS

Our clinical experience with the new absorbable hemostatic bone wax began on March 15, 1948, when the surgeons at the Neurological Institute of New York began the routine use of this material in the operating rooms. All comments by these surgeons regarding its hemostatic properties and ease of handling have been entirely favorable. The surgeons have preferred the new material to the conventional type of bone wax and have used it whenever it was available.

The first 100 consecutive operations in which the new material was employed are tabulated in Table IV. The wounds in 98 of this group developed no infections and healed per primam. In one case, (U. H. 919513) following a laminectomy, a small, superficial abscess developed around a black silk suture. The wound healed promptly after the removal of the suture. In the

other case (U. H. 919223) a large, left fronto-parietal brain abscess was evacuated and the abscess cavity lined with China silk and packed with gauze. This wound continued to drain for a number of weeks.

Out of this group of 100 operations, it was possible to detect significant amounts of fluid under the scalp or in the wound in seven cases. In two cases the fluid was absorbed spontaneously and did not require tapping. Two cases were tapped only once (U. H. 904703) following a bilateral topectomy and (U. H. 890677) following a craniotomy and lysis of Pacchionian granulations. The remaining three patients required repeated taps. One patient (U. H. 725805) had a suboccipital craniectomy with the subtotal removal of a cystic

TABLE IV.—*Clinical Experience With New Absorbable Bone Wax**

Differential section of trigeminal nerve	11
Pre-frontal leukotomy	7
Trephination of skull	5
Craniotomy	39
Sub-occipital craniectomy	4
Topectomy	6
Laminectomy	16
Interlaminar removal of herniated nucleus pulposus	12
Total	100

*Since this article was submitted for publication the new bone wax has been used in a considerable number of general surgical cases. Limited quantity has delayed general trial, but more material will be available as soon as the optimum consistency has been determined.

astrocytoma of the cerebellum. The other two patients (U. H. 897548) and (U. H. 910605) each had the removal of a large meningioma which was then followed by a tantalum cranioplasty. We do not feel that the new absorbable bone wax caused the production of fluid in any of these cases, but believe, rather, that the fluid was a result of the operative procedure itself. In support of this view is the observation that in a number of other cases as many as three tubes of the material, each tube containing three grams of absorbable bone wax, were used following which the wound healed per primam and no fluid collected. Further records were kept but have not been analyzed as the wax has been used routinely.

SUMMARY

A new absorbable hemostatic bone wax has been produced which contains oxidized cellulose as its active component and a mixture of polyethylene glycols of high molecular weight as the vehicle. This material has been found to be actively hemostatic when employed to control hemorrhage from the bleeding surfaces of bone and can be handled and applied more easily than ordinary bone wax.

Laboratory tests with rats indicated that the new material is much less irritating to the tissues than ordinary bone wax and is completely absorbed in three hours.

In experimental rib fractures in dogs both the new material and ordinary bone wax caused some delay in healing. It is not felt that this delay is of any practical significance, however, since the material will not be used in accidental fractures, where early bone repair is the first consideration, nor in weight-bearing bones. In any event, the delay does not seem to be greater than that caused by ordinary bone wax which has been used for the same purpose for over 50 years.

The use of new absorbable hemostatic bone wax in the first 100 clinical neurosurgical operations at the Neurological Institute of New York has been reported. No ill effects or complications which could be attributed to the material have been noted.

Note: The authors wish to express their appreciation to Miss Daisy Mapes, R. N. under whose supervision the animal experiments were carried out.

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FIBROMYXOMA OF THE MANDIBLE*

REPORT OF TWO CASES

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FIBROMYXOMA OF THE FACIAL BONES is a rare and benign tumor. Two patients with this lesion were recently observed at the Hartford Hospital, and their case reports are presented to emphasize certain aspects of the behavior of this non-malignant growth.

Case 1.—E. M., a 32-year-old colored housewife, admitted May 30, 1949, complained of a swelling of the right jaw, the onset of which dated back 3 years and followed extraction of a right lower molar. Subsequently, this swelling was intermittent and essentially asymptomatic. Three months before admission, the remaining two right lower molars were extracted on the advice of the patient's dentist, who felt that infection (not proved) from these roots was responsible for the swelling. Swelling persisted, with occasional bleeding from the site of extraction, up to time of admission, which was precipitated by an upper gastro-intestinal tract upset, non-specific in nature.

The past history was non-contributory.

Pertinent physical findings were confined to the head and neck, where facial asymmetry was noted. This was due to oval swelling of tissues measuring 5 by 4 cm. over the right lower mandible (Fig. 1). The overlying skin was intact. There were no palpable cervical nodes. Intra-oral examination revealed a bulging mass, with ulceration of soft tissue, overlying the alveolar ridge of the horizontal ramus of the right mandible. The mass measured 5 cm. by 4 cm.

Laboratory Data. Blood count, urinalysis and Mazzini tests were negative. There was no abnormality of pulmonary fields on a chest film. Roentgen ray examination of right mandible (Fig. 3) revealed a large soft tissue mass, devoid of calcium, with irregularity of the alveolar process due to bone rarefaction and destruction.

Operation. A biopsy of the mass on June 1, 1949, was interpreted as fibromyxoma. Operation was performed June 7, 1949: A 7 cm. segment of the horizontal ramus of the right mandible (Figs. 5, 6) which was resected after exploration, confirmed by microscopic examination, revealed extensive infiltration by neoplastic tissue of the underlying mandible. Complementary tracheotomy and fixation of mandible by interdental wiring completed the procedure.

Histopathologic Examination. The specimen was reported as "Fibromyxoma of mandible with infiltration of underlying bone" (Fig. 4).

The postoperative course was uneventful. The tracheotomy tube was removed on sixth postoperative day. The wounds healed per primum and the patient was discharged on fourteenth postoperative day with interdental splints in place. The cosmetic and functional result was excellent (Fig. 2).

Case 2.—V. L., a 22-year-old white married male was admitted to the hospital July 4, 1949, with a huge mass protruding from the right malar area. At the age of 17, 5 years before this admission, he received 3 radium treatments for a small mass in the right hard palate, which had been biopsied and reported "fibromyxoma." Patient failed to return for further therapy and follow-up.

* Submitted for publication October, 1949.

The mass continued to grow progressively, with marked acceleration 6 months before admission. Local pain, headache, bleeding from the hard palate, loss of teeth from the right upper alveolar ridge, and restriction of diet to liquids and soft foods combined with the marked facial deformity prompted admission to the hospital.

The past history, familial background and review of systems were non-contributory.

Positive physical findings were confined to the head and neck where a large round swelling 10 cm. in diameter (Fig. 7) overlay the right malar area, elevated the right orbit superiorly, obliterated the right naso-malar fold, displaced the nose medially, tautly stretched the right upper lip, and raised the right zygoma laterally and posteriorly. The overlying skin was thinned out, and erythematous, but intact. There were no palpable cervical nodes. On intra-oral examination, a large ulcerating, pink-grey, friable, vascular solid mass replaced the entire right hard palate and extended over the mid-line to involve all but the lateral 2 cm. of the upper left alveolar ridge. The clinical impression was recurrent fibromyxoma.

Laboratory Data. The blood count, urinalysis, and serology were essentially normal. Stereoscopic films of the facial bones (Fig. 9) demonstrated a large soft tissue mass destroying the right facial bones including the floor of orbit, alveolar process, zygoma and extending into the right ethmoid cells. The base of skull did not appear eroded.

Operation. On July 7, 1949, a bilateral ligation of the external carotid arteries (above superior thyroid) was followed by a radical resection "en bloc" of the right maxilla and ethmoids (Fig. 10). Frozen section microscopic examination was reported fibromyxoma. A split-thickness anterior abdominal wall dermatome graft was applied to the raw surface of the right malar cheek flap to minimize contracture of this tissue. Following removal of the tumor, support for the orbit was absent. Exenteration of this orbit hardly seemed desirable in view of the youth of the patient and benignity of the tumor. Therefore, the tendon of the right temporalis muscle was divided at its tendinous insertion into the right mandible and this was brought medially and sutured to the nasal bones as a sling for the right eye. The entire procedure was tolerated satisfactorily.

The postoperative course was uneventful. Nutrition was maintained by nasal tube feedings. The intra-oral pack was removed on seventh postoperative day. Sixty per cent take of the graft was noted. Temporal muscle flap support for the right orbit was excellent and vision satisfactory. Histopathologic examination was reported as "fibromyxoma" (Fig. 11).

At operation on July 1, 1949, a rim of 1 cm. of the residual left hard palate was removed, since myxomatous tissue was observed on the bony edge postoperatively. Adequate surgical margin then obtained and confirmed by microscopic examination.

The defect healed satisfactorily (Fig. 8). A temporary sponge prosthesis was used to fill this defect, to improve speech and allow the patient to eat. It is planned to fit an acrylic prosthesis when maximum contracture has occurred.*

DISCUSSION

In the absence of etiologic information or experimental investigation regarding this tumor, any discussion as to its origin and natural life history is of necessity controversial. Furthermore, there are too few reports[1, 2, 5-7] in the medical literature to permit rational evaluation of choice of therapy and end results.

Ewing[3] points out that many mesoblastic tumors, such as fibromata, lipomata, etc., often undergo myxomatous change under the influence of chronic irritation or degenerative change from diminished blood supply. Willis[4] essen-

* Last follow-up: June, 1950, revealed no evidence of recurrence. Vision was satisfactory in right orbit. Acrylic prosthesis was completed and being used to satisfaction of patient.

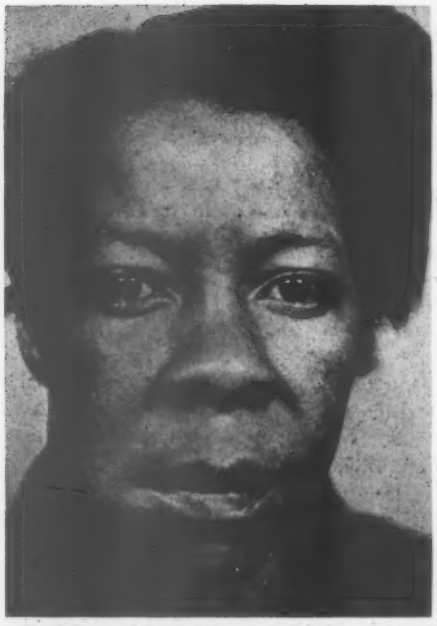

FIG. 1

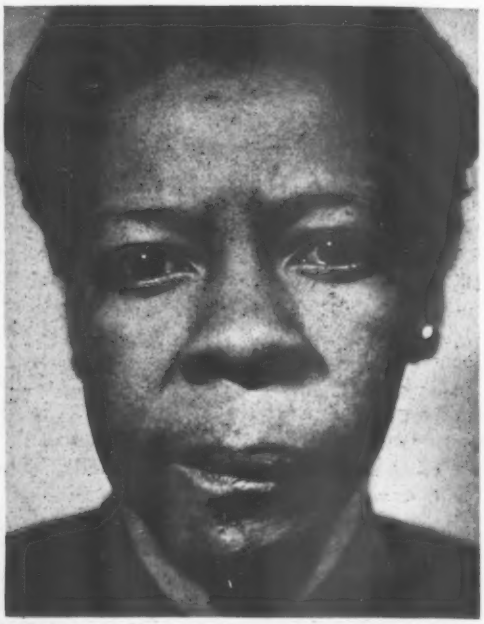

FIG. 2

FIG. 3

FIG. 4

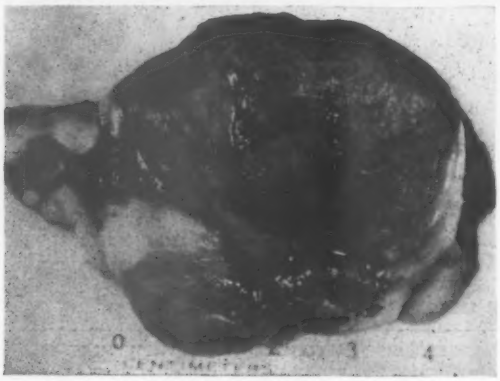

FIG. 5

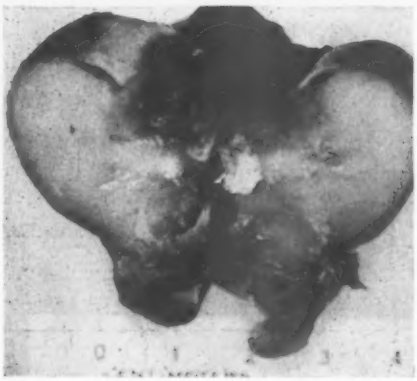

FIG. 6

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tially subscribes to this concept with the additional observation that this group of tumors probably arises from the nerve sheath. Thoma and Goldman[5] suggest that myxomatous degeneration of odontogenic fibroma arising from the mesenchymal portion of the tooth germ account[3] for this tumor in the upper and lower mandibles.

FIG. 7

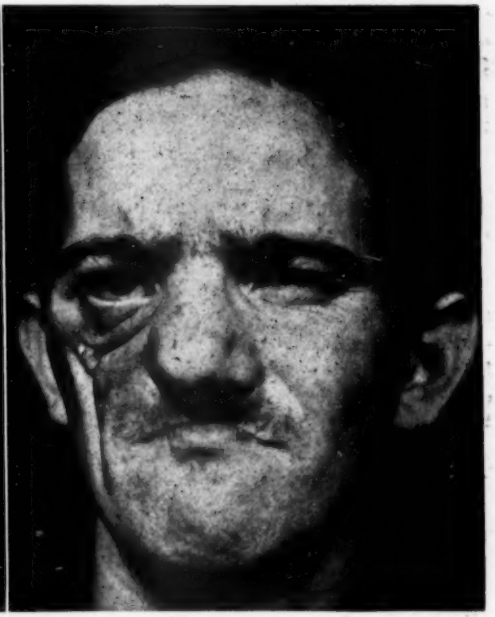


FIG. 8

FIG. 7.—Case V. L. Preoperative photograph demonstrating marked facial asymmetry of right maxillary tumor.

FIG. 8.—Case V. L. Photograph ten weeks after operation. Vision in right eye intact. Despite split-thickness graft lining undersurface of right cheek flap, contracture of this skin resulted (probably due to marked stretching of this skin by tumor).

Clinical experience indicates that these tumors can attain large size, are often poorly encapsulated, and are of a homogeneously firm multi-lobular consistency. Cut section reveals a translucent and gelatinous surface. Since encapsulation may be incomplete, infiltration of surrounding tissues, and

FIG. 1.—Case E. M. Frontal view of patient with fibromyxoma of right mandible. Note swelling of soft tissues and facial asymmetry.

FIG. 2.—Case E. M. Postoperative photograph, four months after surgery.

FIG. 3.—Case E. M. Roentgenogram of right mandible demonstrating erosion of superior alveolar ridge. Infiltration of mandible below this area of bone absorption required resection of this segment of mandible.

FIG. 4.—Case E. M. Fibromyxoma of mandible. Tumor composed of loosely arranged fibrous tissue of adult appearance with replacement of bone. In lower part of photomicrograph a small fragment of mandible may be seen. (H and E x 70).

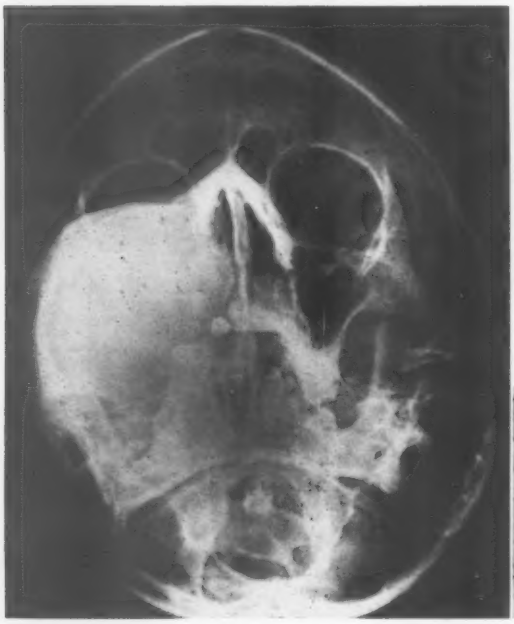
FIG. 5.—Case E. M. Resected horizontal ramus of right mandible. The encapsulated and ulcerated oral surface of this lesion is demonstrated.

FIG. 6.—Case E. M. Cut section of fibromyxoma demonstrating partial encapsulation of superior intraoral surface and infiltration of inferior mandibular segment.

recurrence logically follow a limited enucleation of this lesion. However, when these tumors are small, they may be well circumscribed, noninfiltrating and thus amenable to local excision. Ultimate decision as to the extent or operation must rest upon careful correlation of preoperative roentgen rays, gross appearance of tumor at operating table, and frozen section microscopic study of surgical margins.

Both of our cases demonstrate the infiltrative nature of this growth. The first patient had involvement of virtually the entire segment of the underlying mandible, requiring segmental resection of this structure. The second patient

FIG. 10



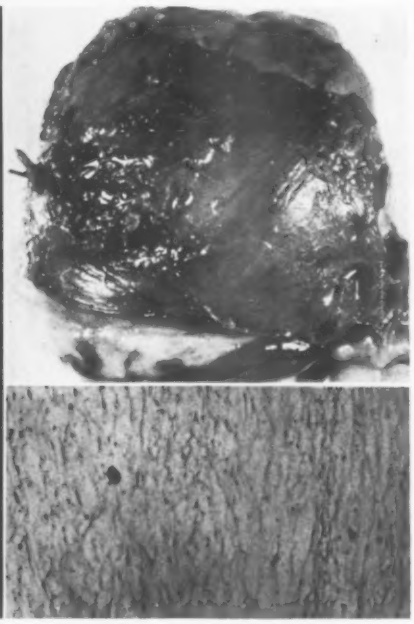


FIG. 9 FIG. 11

FIG. 9.—Case V. L. Roentgenogram of facial bones reveals expansile character of this lesion with destruction of bony walls of right maxilla.

FIG. 10.—Case V. L. Operative specimen. Lateral view. Note encapsulation without evidence of bony parietes of maxilla.

FIG. 11.—Case V. L. Fibrous myxoma of maxilla. Loosely arranged fibrous tissue cells forming a recticular pattern. (H and E x 55.)

presented complete replacement and absorption of all walls of the right maxilla, including infra-orbital plate, as well as extensive infiltration into the opposite half of the hard palate. (Histopathologic studies of several cases documented in the literature fail to mention this characteristic.) It should be pointed out that mixed tumors of salivary glands which often contain fibromyxomatous tissue frequently present similar characteristics of poor encapsulation, and tendency for recurrence following enucleation or limited excision.

The large size of the fibromyxoma, pressure on surrounding vital structures, together with cosmetic deformity, often prompt treatment for this benign tumor. Since this growth may infiltrate bone, and is composed of well differentiated tissue, it is unlikely that radiation therapy would be effective in eradicating the lesion. Therefore, surgical resection, with microscopic control to determine adequate margin, logically suggests itself. Whether incomplete removal, followed by repeated recurrence, favors subsequent malignant degeneration is not apparent from the limited follow-up available in the small series reported in the literature.

It is of some interest to note, however, that this tumor is commonly confused with the benign dentigerous cyst, and indeed, diagnosis is often made only following biopsy. Furthermore, the majority of patients presenting this growth are in their second and third decades.

SUMMARY

Fibromyxoma of the facial bones is a benign tumor of infrequent occurrence. Both of our patients presented infiltration and destruction of bone. From the limited number of case reports available, it appears to have occurred in young adults and most frequently simulates the dentigerous cyst. Treatment of choice logically appears to be adequate surgical resection with careful microscopic control. Prosthetic and plastic reconstruction is a variable adaptable to the individual case.

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CHOLEDOCHUS CYST

A CASE TREATED BY Y-ROUX TYPE ANASTOMOSIS OF JEJUNUM TO THE CYST*

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THE MAJORITY OF PATIENTS with cystic dilatation of the common bile duct who have recovered have been treated by primary anastomosis of the biliary system and the gastro-intestinal tract. In the patient treated by the authors this was accomplished by a "Y" or Roux-type anastomosis. As this method of treatment seems to the authors to involve possible advantages, a description of the case and a discussion of the rationale is hereby presented.

In their publications, Shallow, Eger and Wagner[1, 2] have reviewed and discussed 182 cases. They conclude that the safest treatment, and the one recommended for general employment, is anastomosis of the cyst to the duodenum. They mention the possibility that, with this treatment, the remaining duct may harbor regurgitated food with consequent severe ascending cholangitis. Pearce *et al.*[3] have shown that in dogs with a Y-type anastomosis an antiperistaltic loop of bowel 12 inches or more in length will prevent regurgitation of intestinal contents. It therefore seems to the authors that the method of treatment that they have utilized in a case of choledochus cyst, in which such a loop was used, might serve to diminish one of the disadvantages of what would otherwise be the preferred method.

CASE REPORT

B. W. was a 17-year-old white female. According to her father, the patient had suffered several severe attacks of colicky abdominal pains as an infant. The last of these attacks was accompanied by jaundice for which she was hospitalized.

The following notes are taken from a summary of the record of B. W. which Dr. Paul V. Woolley, Jr., of Children's Hospital of Detroit, has very kindly provided. The patient first entered the hospital when she was 16 months old, on October 6, 1933, with a complaint of attacks of jaundice during the previous six months. Examination revealed an enlarged liver and a distended, enlarged gallbladder. In an attempt to roentgen ray the gallbladder, after giving "Iodeikon" by mouth, nothing was visualized in the plate. The child was kept on a low fat diet during her entire stay in the hospital. She developed a cold and the middle ears had to be opened; they drained pus and the child was acutely ill for several days. She lost considerable weight. She did not have another attack of jaundice, however, and it was thought best to discharge her and have her return if there was another attack of jaundice. She was discharged on October 26, 1933. The diagnosis was chronic cholecystitis, chronic cholangitis and chronic hepatitis.

Following this episode the patient showed no further jaundice and she is said to have remained perfectly well until the present attack, 15 years later. The present attack

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began on May 27, 1949, with a severe upper abdominal cramp, nausea and vomiting. This cleared up, and for the next two days the patient felt well. On May 29, she again experienced severe upper abdominal pain with severe upper abdominal cramps and nausea and vomiting. An enema was given at home, but the patient got no relief and she was admitted to Providence Hospital, Detroit.

Examination showed no abdominal spasm, rigidity or tenderness. A questionable upper abdominal mass could be felt, but did not reveal itself fully until the patient was completely relaxed under anesthesia. On admission, the temperature was 98.2°F, the pulse rate 80, and the respiratory rate 20. Laboratory findings were as follows: Hemaglobin, 16.6; R.B.C. 5,500,000; white cells 15,400; differential: 97 per cent neutrophils,

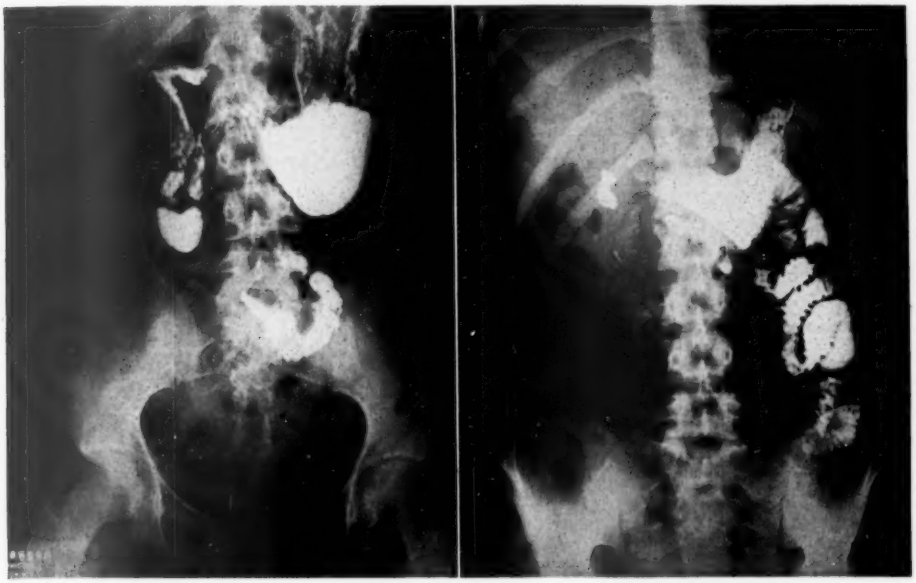

FIG. 1 FIG. 2

FIG. 1.—Four and one-half months postoperatively. This first view shows the descending limb of the duodenum to be somewhat elongated, and a minimal delay was suggested in the transverse portion of the duodenum. No irregularities or obstruction could be noted. The spill into the jejunum was followed readily, with the barium advancing rather slowly. At no time during this examination could barium be noted extending toward the biliary tract region. The passage of the barium through the upper small bowel appeared entirely normal.

FIG. 2.—A final study was made one-half hour later and demonstrates a normal advance of the barium through the left small bowel, with again no evidence of any barium extending up toward the biliary tract.

3 per cent lymphocytes (2 per cent non-filamented forms). The urine analysis was negative for sugar and albumin. The preoperative diagnosis was inflammation of an abnormally located appendix or possible cholecystitis. The patient was taken to the operating room.

A right rectus incision was made. Examination of the appendix and the pelvic organs showed these structures to be normal. There was a yellowish-green discoloration of the gastro-colic ligament as if there had been leakage of bile. Exploration of the right upper quadrant of the abdomen revealed a large firm mass. Careful examination of this mass showed it to be retro-duodenal and the first and second portions of the duodenum

were incorporated in the mass. The second portion of the duodenum was stretched out on the lateral side of the mass. The foramen of Winslow was obliterated. The gallbladder, which was small and surrounded with adhesions, was displaced upward.

The duodenum was mobilized by the Kocher method. This maneuver made possible a full exposure of the mass. It was seen to extend from the porta hepatis, above, to a point behind the head of the pancreas, below. Aspiration of the cystic mass yielded approximately 800 cc. of clear light green bile. A 4 or 5 cm. incision was then made into the anterior border of what appeared to be the common duct. Exploration with the index finger showed that this cystic dilatation extended all the way up into the liver, while caudally it extended as low as the third part of the duodenum and posterior to the head of the pancreas.

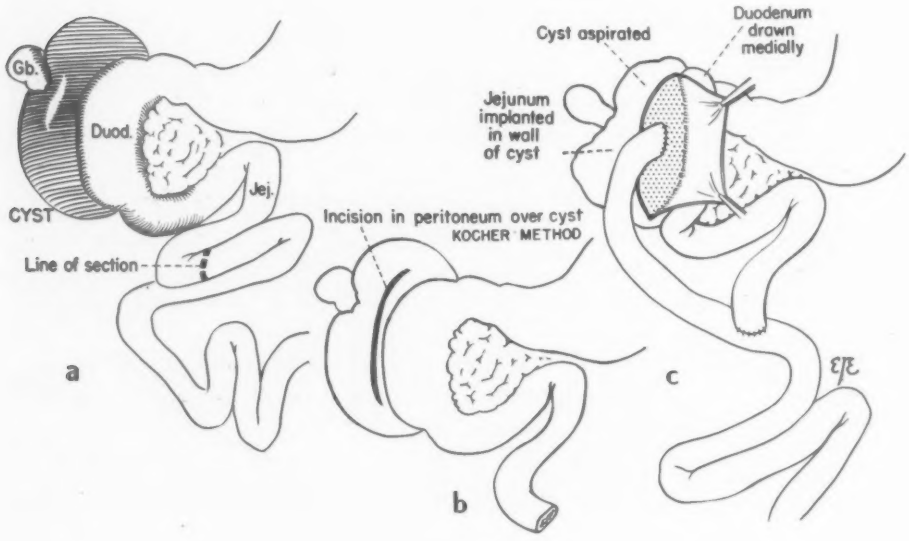

FIG. 3

An opening was then made into the avascular portion of the transverse mesocolon through which a loop of jejunum was brought. The mesocolon was sutured around this loop. A Y or Roux type of anastomosis was then made to the common duct cyst. The end of the distal limb of jejunum was anastomosed to the side of the cystic dilatation with interrupted mattress sutures. Anteriorly a row of Connell's stitches approximated the serosal surfaces of the bowel and the cyst wall. The end of the proximal limb was then anastomosed to the side of the distal limb of jejunum 12 inches distal to the first anastomosis. This was also done with interrupted chromic sutures. The omentum was brought up to both of these anastomoses. A Penrose drain was inserted into the right paracolic fossa and brought through the lowermost part of the incision. After inserting three interrupted silk sutures, the abdomen was closed.

Convalescence in this case was rather stormy. The patient continued to vomit and in spite of parenteral proteins, vitamins, glucose, electrolytes, and chemotherapy she suffered dehiscence of her wound on June 7, 1949. This was repaired and at the time evidence of localized peritonitis about the cyst was noted. Despite continued penicillin and streptomycin therapy there was no marked improvement during the next five days. At

the end of that time she was given sulfadiazene intravenously. Following this her recovery was uneventful and she was discharged from the hospital on June 23, 1949.

Four and one-half months later the patient had regained most of the weight said to have been lost during her illness. She stated that she felt "perfectly well" and she denied recurrence of any of the symptoms.

A radiologic examination was undertaken by Dr. W. A. Irwin. Gallbladder dye was administered by mouth but roentgen ray studies failed to reveal any concentration.

Following a barium meal fluoroscopic examination and a series of 4 radiograms failed to show any extension of barium toward the biliary tract even after pressure on the abdomen. The descending loop of the duodenum appeared somewhat elongated. The passage of barium through the upper small intestine was somewhat slower than usual but otherwise findings were reported to be entirely normal.

DISCUSSION

Choledochus cysts are rarely encountered. Shallow *et al.* were able to find only 182 cases in the literature up to 1946.[1, 2] Little can be added to their summary concerning etiology or diagnosis. It may be pointed out, however, that in this case the patient had had jaundice 15 years previously when 16 months old. In children with jaundice or colicky abdominal pains, choledochus cyst is, therefore, one of the possibilities and study of the gallbladder duct system should be considered.

From the standpoint of treatment, the present case emphasizes several considerations.

1. In operations for choledochus cyst, the Kocher maneuver may be of considerable assistance. In the present case the technic simplified the procedure and permitted easier access.

2. Shallow *et al.*[2] recommend the anastomosis of the cyst to the duodenum, but point out the risk of ascending cholangitis from regurgitation of food. The alternative of extirpation of the cyst followed by primary anastomosis of the remainder of the biliary duct system to the duodenum reduces this risk, but Shallow and his colleagues indicate a higher mortality and recommend the method only when the patient is a good operative risk, the cyst large and infection minimal or absent.

3. Michel[4] also mentions the possibility of reflux into the cyst and claims that this can be eliminated by the Braun type of anastomosis as demonstrated in his case. In fluoroscopic studies he was unable to force barium into the cyst even by pressure from the outside, although it did enter the anastomotic loop of intestine.

4. Tsujimura[5] and Kambe[6] reported one successful operation each in which they performed a Y-type anastomosis between the jejunum and the cyst. Fujihara[7] reports a third such case which he successfully treated. He severed the jejunum 50 cm. analwards from the ligament of Treitz and, after closing both stumps, performed a side-to-side anastomosis of the distal stump with the cyst and an anastomosis of the side of the proximal stump with the side of the distal limb at a point 30 cm. analward from the previous anastomosis. In discussing operative methods for anastomosis of a choledochus cyst with the

gastro-intestinal tract, Fujihara comments that a Y-form anastomosis using a long by-passed limb of jejunum, as in his case, is the ideal form and should be considered the normal method of treatment.

5. Although Michel[4] cites abstracts of the reports on these Japanese cases, he considers the Y-type anastomosis more dangerous than the method he used. Thus the advantages of the Y-type anastomosis, although possibly discussed in the original Japanese publications, do not appear to have been heretofore mentioned in the European or American literature on choledochus cyst.

SUMMARY

The treatment with a Y-Roux type anastomosis, with an antiperistaltic limb of jejunum of adequate length anastomosed to the cyst, would tend to eliminate one of the disadvantages of anastomosis of a choledochus cyst to the duodenum and would avoid the risks of extirpation of the cyst.

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ENTEROGENOUS CYST OF THE DUODENUM*

A CASE REPORT

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Because of its rarity, a case of enterogenous cyst of the duodenum is of sufficient interest to be reported. We have found only 18 cases previously reported in the literature.[1-18]

CASE REPORT

L. D., a 30-year-old white female, was admitted to the Lincoln Hospital on December 20, 1948, complaining of generalized abdominal pain for 3 days. She had been nauseated during this time but had vomited only once, on the morning of admission, after which the pain shifted to the right lower quadrant of the abdomen. For the past 2 years there had been attacks of mild indigestion.

Physical Examination. The patient was well-nourished and fairly comfortable. The temperature was 101.8 F.; pulse, 116; respiration, 24; blood pressure, 100/60. The positive findings were limited to the abdomen. There was moderate tenderness, rebound tenderness and slight muscle guarding in the right lower quadrant. The white blood count was 10,000; 90 per cent polymorphonuclear leukocytes, 2 per cent bands, 7 per cent lymphocytes, and 1 per cent monocytes. Hemoglobin, 12.9 Gm. and red blood cells, 3,600,000. The urinalysis was negative. A preoperative diagnosis of acute appendicitis was made.

Operation. Through a McBurney incision, the abdominal cavity was found to contain a moderate amount of clear serous fluid. The appendix was small and atrophic. A Weir extension was made and a small ruptured Graafian follicule was visualized in the right ovary. However, on further exploration, a mass was palpated in the right gutter retroperitoneally. The McBurney incision was then extended cephalad to the costal margin, cutting across the oblique and transverses muscles, to obtain better exposure. The lateral peritoneal fold of the right colon was incised, and by blunt dissection, the mass was exposed behind the right colon extending up to the level of the hepatic flexure. The mass was one and a half times the size of a normal kidney and felt cystic. There was marked edema of the tissues about it. The right kidney was palpated above and behind the mass. The stomach was visualized and traced to the pylorus. It then became evident that the mass was a tremendously dilated second portion of the duodenum. A polypoid, cystic mass, oval in shape and mobile, could easily be palpated through the wall of the second portion of the duodenum. The gallbladder appeared slightly thickened and the common duct was slightly dilated, but there were no stones palpable in either of these structures. The pancreas felt indurated and nodular. The area was packed off and the second portion of the duodenum was opened through a 6 cm. longitudinal incision in its anterior wall. A large polypoid cystic mass measuring 4 cm. wide by 8 cm. long was found within the lumen of the duodenum, attached to the posterior wall of the duodenum (Fig. 1). It could not be emptied by compression. The wall of the cyst was congested and presented a small area of gangrene at its fundus. There was a pin head sized opening at the middle of its right side which drained bile. When probed, this was seen to be the termination of the common bile duct. The cyst was opened at its fundus and was found to be filled with bile.

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There were about two dozen facetted gall stones in the lumen of the cyst. Duodenal mucosa appeared to line both the inside and outside of the cyst. The pancreatic ducts were not found, nor was the opening from the common bile duct directly into the cyst visualized. Most of the cyst wall was excised, except the portion containing the termination of the common bile duct, and the small remaining portion of the cyst wall was sutured with interrupted 00 chromic. The common bile duct was then opened above the duodenum and a T tube inserted for decompression. The duodenotomy was then closed with two layers of sutures. Three drains were inserted in the retroperitoneal area and the abdomen closed in layers.

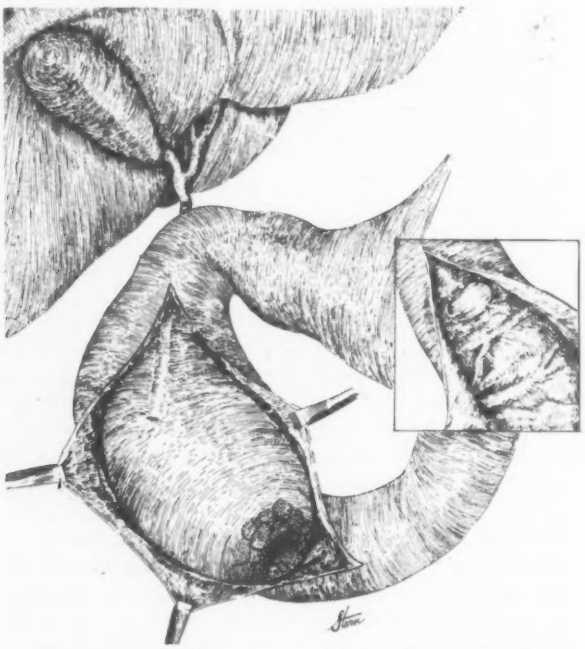

FIG. 1.—Artist's sketch of the findings at operation. The duodenum is open, revealing the cyst arising from the posterior wall, with the common bile duct on its anterior surface, and containing gall stones. Insert reveals the appearance after excision of the cyst wall.

The patient made a completely uneventful recovery. Cholangiograms taken on the twelfth postoperative day showed easy passage of the diodrast into the duodenum, and the T tube was removed on the fifteenth day. Gastro-intestinal roentgen ray series at this time showed only slight dilatation of the second portion of the duodenum. When seen 20 weeks after discharge from the hospital, the patient was asymptomatic and had gained eight pounds in weight.

Microscopic examination of the cyst wall revealed it to be lined on both the inside and outside with typical and identical duodenal mucosa (Fig. 2). This has been the case in all enterogenous cysts of the duodenum in which the microscopic appearance has been reported. Separating the mucosal layers was a thin muscularis mucosae. Thus this cyst was of the submucous variety.

Enterogenous cysts are always lined with intestinal mucosa, although it does not always correspond to the mucosa of the intestine adjacent to the

cyst. The fluid contained within these cysts has been clear and serous with the exception of the three that were located at the ampulla of Vater and contained bile.[12, 14] This case is the only one reported to contain gall stones. The cysts may be attached to any portion of the duodenal wall. The majority were attached to the posterior wall, especially those in the second portion of the duodenum. They may be of the submucous, intermuscular or of the subserous variety.

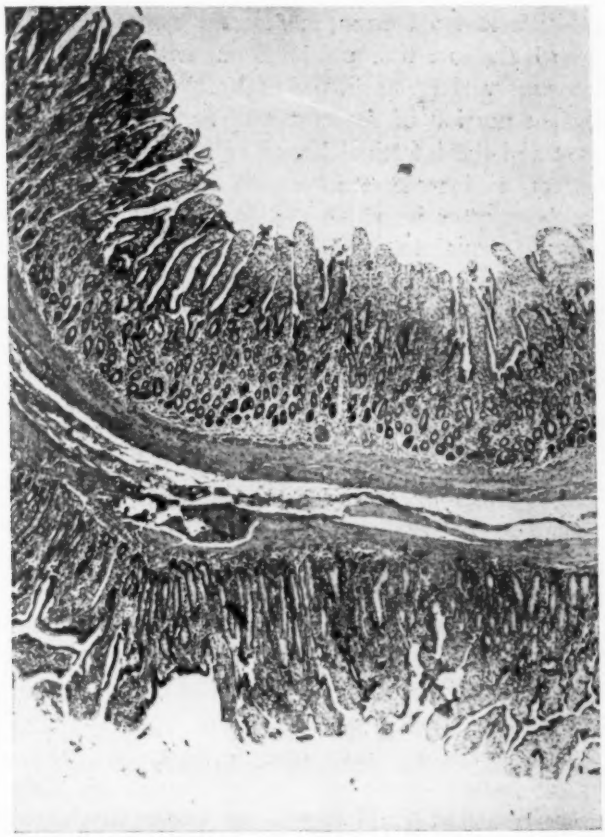

FIG. 2.—Photomicrograph revealing the wall of the cyst, lined on either side by duodenal mucosa with a thin layer of muscularis mucosae separating the mucosal layers. (Harold H. Briller, M.B.P.A., New York, N. Y.)

Enterogenous cysts of the duodenum may remain dormant for long periods of time and produce no symptoms. On the other hand, they may produce symptoms of obstruction, pain, and a palpable epigastric or right upper quadrant mass from time of birth. Those cases which are seen in infancy are usually diagnosed as hypertrophic pyloric stenosis. Ten cases[1, 5, 7, 9, 11, 15] have been reported in patients under four months of age. Of these, two[1, 2] were in newborn; one[3] was not operated upon and died 30 days after admis-

sion to the hospital; four[4, 5, 7, 10] were operated upon and died shortly thereafter; and three[9, 11, 15] were operated upon and recovered—presenting a mortality of 70 per cent in this group. In sharp contrast is the group of nine patients, ranging in age from four and one-half to 69 years, who were operated upon with no mortality.

The treatment of choice is resection of the cyst with or without adjacent duodenum. It is sometimes impossible to remove the cyst in its entirety without resecting a portion of the duodenal wall, and for this reason, enterogenous cysts must be differentiated from mesenteric cysts, which do not have a common wall with the intestine and shell out without difficulty. If the cyst is of the submucous variety, as in the authors' case, it is necessary only to remove a sufficient portion of the cyst wall to establish free communication between the cyst and the intestinal lumen to insure adequate drainage. This produces, in effect, an internal anastomosis between the cyst cavity and the duodenum. In those cases in which the cyst can be dissected out, one must be certain that the remaining duodenal wall retains sufficient strength to withstand intraduodenal pressure. If resection or internal anastomosis is not feasible, gastroenterostomy will relieve the obstruction and may achieve a satisfactory result.[9] Complete or partial marsupialization was carried out in three cases but was attended by 100 per cent mortality.[4, 7, 10]

SUMMARY AND CONCLUSIONS

1. The nineteenth case of enterogenous cyst of the duodenum, and the only one containing gall stones, is reported.

2. The salient features of those cases which have been reported in the literature are reviewed.

3. The prognosis depends upon the age of the patient at the time that symptoms necessitate surgical intervention.

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ANNOUNCEMENT OF VAN METER PRIZE AWARD

The American Goiter Association again offers the Van Meter Prize Award of Three Hundred Dollars and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The Award will be made at the annual meeting of the Association which will be held in Columbus, Ohio, May 24, 25 and 26, 1951, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English; and a typewritten double spaced copy in duplicate sent to the Corresponding Secretary, Dr. George C. Shivers, 100 East Saint Vrain Street, Colorado Springs, Colorado, not later than March 1, 1951. The committee who will review the manuscripts is composed of men well qualified to judge the merits of the competing essays.

A place will be reserved on the program of the annual meeting for presentation of the Prize Award Essay by the author, if it is possible for him to attend. The essay will be published in the annual Proceedings of the Association.

NEUROGENIC TUMOR OF THE STOMACH*

CASE REPORT

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NEUROGENIC TUMORS of the stomach are not common and they are difficult to classify pathologically. Most reports in the literature are of small series of cases or are single, isolated cases.[1-6, 12, 15] Minnes and Geschickter,[10] in classifying a series of 931 cases of benign gastric tumors, found that 10.9 per cent were of neurogenic origin.

It is not the purpose of this report to discuss the histopathology of neurogenic gastric tumors. There is considerable difference of opinion among pathologists as to the origin and classification of these tumors. They are referred to by many names, such as: neurinoma, neurilemmoma, schwannoma, and perineural fibroblastoma. For discussions on histopathology, the reader is referred to the writings of Mallory,[8] Masson,[9] Stout,[13, 14] and Ransom and Kay.[11]

Clinically, neurogenic stomach tumors behave much the same as other benign gastric neoplasms. Symptoms vary according to the size and location of the tumor. There are often certain characteristics manifested by benign gastric tumors. Probably the most outstanding of these is the frequency with which hemorrhage occurs, and the hemorrhage is often not associated with cachexia and weight loss such as that found in gastric carcinoma. Also, benign gastric neoplasms often possess characteristic roentgenographic patterns. These findings have been shown by Carter and Laing[5] and by Finesilver.[7]

The diagnosis of gastric tumors of neurogenic origin can only be made microscopically. Once the diagnosis is made, the surgeon should keep the following facts in mind regarding these neoplasms: (1) growth is usually slow; (2) malignant change is not common; (3) metastasis is rare; (4) local recurrence following excision is frequent. Obviously, the treatment of choice is adequate surgical excision, and, if this is done, the result will be satisfactory.

The following case has been classified as a neurilemmoma. The clinical picture is typical for this type of tumor although it is of unusually large size. The patient has been followed approximately three years since resection, and there is no evidence of recurrence to date.

CASE REPORT

G. H., a 45-year-old white male, was admitted to the Germantown Hospital on October 9, 1946, complaining of weakness, vague abdominal discomfort and tarry stools.

* Presented before the Philadelphia Academy of Surgery April 5, 1947. Submitted for publication September, 1949.

His history dates back 13 years. During this time, he suffered several severe hemorrhages, presumably gastric in origin. Between 1934 and 1946, he was hospitalized on four occasions for hemorrhage. In 1934, the diagnosis of bleeding peptic ulcer was made, and the patient was treated medically. In 1941, the same diagnosis was made and the same treatment was repeated. In 1942, laparotomy was performed, and the diagnosis of inoperable carcinoma of the fundus of the stomach was made. In 1943, laparotomy was again performed, and a biopsy of the tumor revealed a neurilemmoma. Technically, the tumor could not be resected through the abdominal approach because of adhesions to the spleen and diaphragm. The patient refused further operative intervention, was discharged, and remained in good health until the present admission.

The positive findings on admission were a moderate secondary anemia and roentgen ray evidence of a large tumor in the fundus of the stomach (Fig. 1). The patient was

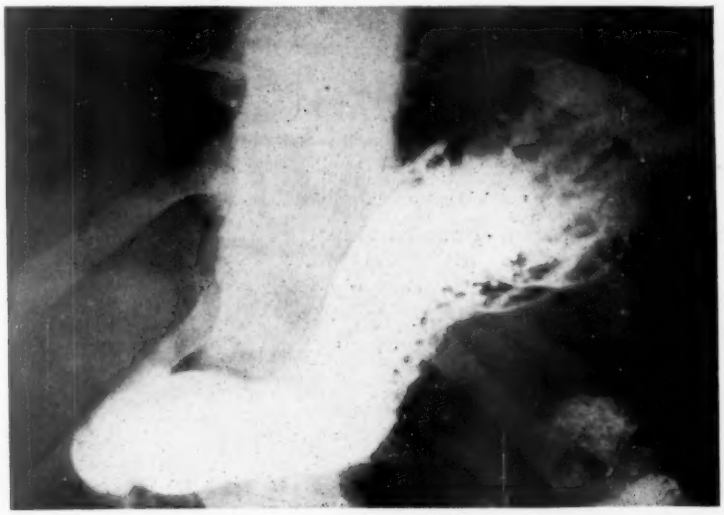

FIG. 1.—Preoperative roentgenogram showing large tumor mass in fundus of stomach.

prepared for operation with transfusions and general supportive measures. Operation was carried out two weeks later under endotracheal ether anesthesia, employing a left transthoracic approach. The tumor mass could be palpated through the diaphragm and was partially adherent to its under surface. Some difficulty and troublesome bleeding was encountered in opening the diaphragm and freeing the tumor. Splenectomy was necessary because the tumor was adherent to this organ. Exploration revealed no evidence of metastasis to the liver or lymph nodes in the area. The tumor was "hour-glass" shaped and appeared to arise from the muscularis (Fig. 2). The smaller portion of the tumor lay within the stomach while the larger portion was entirely outside the stomach wall. It was well encapsulated except for the extragastric portion, which was adherent to the diaphragm and spleen. The intragastric portion of the tumor was covered with intact mucous membrane. It was possible to remove the tumor with a margin of normal stomach by performing a partial fundusectomy. The stomach was closed in layers. The diaphragm and chest wall were closed, and the patient was returned to the ward in good condition.

An uneventful postoperative course followed. The patient was allowed out of bed on the fifth day and was discharged on the fifteenth day. Since discharge, the patient has been symptom-free. Follow-up gastro-intestinal and chest roentgenograms show no evidence of recurrence.

Pathologic Examination. Gross Pathology: The specimen reveals an "hour-glass" shaped tumor arising from the stomach wall. The smaller, or intragastric, portion measures 80 by 60 by 40 mm. and weighs 123 Gm. The surface is covered with essentially normal gastric mucosa. The tumor is firm in consistency and presents multiple cystic areas containing hemorrhage on cut surface. A greater part of the cut surface has a

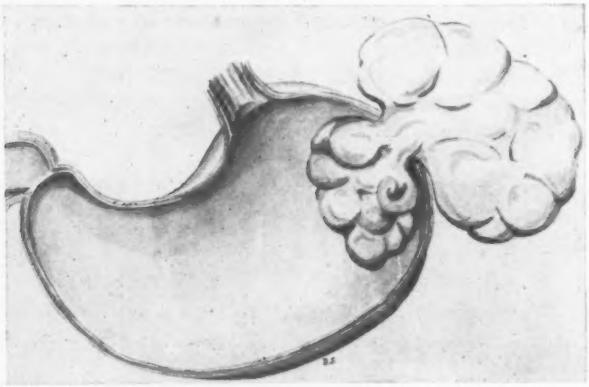

FIG. 2.—Sketch of tumor and its relationship to stomach.

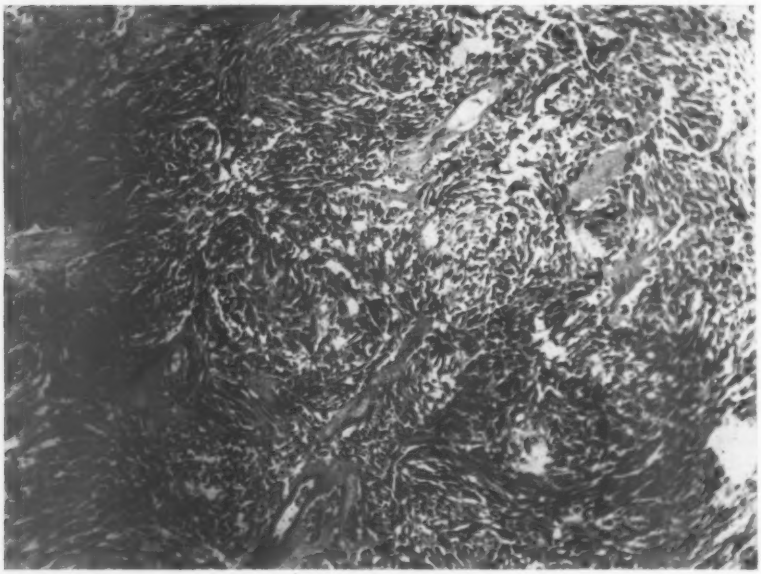

FIG. 3.—Photomicrograph of section of tumor.

yellowish granular appearance except at the periphery, where it is reddish-brown in color. The tumor has a well defined capsule approximately 1 mm. in thickness. The larger, or extragastric, portion of the tumor has similar characteristics. It measures 110 by 80 by 70 mm. and weighs 301 Gm.

Microscopic Examination. Section through the tumor reveals a neoplasm composed of spindle cells with oval nuclei arranged in a palisading manner. There is a large

amount of collagenous intracellular substance. The neoplasm does not involve the mucous membrane and appears to be well encapsulated. There are few mitoses, and there is considerable vascularity (Fig. 3).

Diagnosis. Neurilemmoma.

SUMMARY

A case of neurogenic stomach tumor is presented. Treatment was accomplished by means of surgical resection employing the transthoracic approach. The patient is alive and free of symptoms approximately three years postoperatively.

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ANNALS OF SURGERY

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SOLITARY PYOGENIC ABSCESS OF THE LEFT LOBE OF THE LIVER

A REPORT OF TWO CASES*

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FROM THE VETERANS ADMINISTRATION HOSPITAL, DALLAS

While solitary pyogenic abscesses of the liver occur infrequently, involvement of the left lobe alone is even more rare. In large series, abscesses of the left lobe as compared with the right occur in a ratio of one to eight or nine.[1]

Two cases are reported here which involved the left lobe of the liver and were primary pyogenic abscesses:

Case 1.—*Solitary pyogenic abscess of the left lobe of the liver drained in two stages.* TGR, a white man, 40 years of age, was admitted to the Veterans Administration Hospital, Dallas, Texas, November 15, 1947, complaining of left upper quadrant abdominal pain. Two weeks before admission he experienced a moderately severe pain in the left upper quadrant of the abdomen near the midline, five hours after eating a light lunch. The pain consisted of cramps occurring every 15 minutes, lasting 30 to 60 seconds, with complete freedom from pain between the cramps. The attack lasted more than an hour. He took a large dose of epsom salts, but had no relief of the discomfort. However, he experienced complete relief after copious defecation initiated by an enema. Five days later, about 8 o'clock in the evening, approximately six and a half hours after a noon meal, he suffered an attack similar to the previous one. His temperature at this time rose to 104° F. He felt weak. The pain was cramping, occurring every 10–15 minutes in the left upper quadrant near the midline. He was given 2 doses of penicillin intramuscularly by his physician, and this appeared to relieve his fever. From that time on he felt weak, had no appetite, and had recurrences of the cramping abdominal pain. The pains were not as severe as with the original attack. The discomfort remained persistently in the left upper quadrant of the abdomen. In addition he experienced chills and fever and noted a mass in the left upper quadrant.

He appeared moderately uncomfortable with a temperature of 101.6° F. There was the suggestion of a mass, the size of a grapefruit, in the epigastrium extending to the left hypochondrium. There was no abdominal tenderness. A tentative diagnosis was made of an ulcer on the anterior surface of the stomach, which had perforated, with walling off by the omentum, or an abscess of the left lobe of the liver. Roentgenograms of the chest and abdomen appeared negative. Roentgen examination of the gastro-intestinal tract indicated external pressure on the stomach by a mass in the left upper quadrant, probabiy due to enlargement of the left lobe of the liver, although a cyst or tumor of the tail of the pancreas could not be excluded (see Fig. 1). Examination of the feces revealed no ova or parasites. Serum amylase was normal. He had a moderate leukocytosis. All agglutinations were negative. There was evidence of mild liver damage with 4 plus thymol turbidity, 2 plus cephalin flocculation, and prothrombin time of 75 per cent of normal.

Preoperatively he was given 50,000 units of penicillin intramuscularly every 3 hours without marked improvement. On December 4, 1947, under general anesthesia, exploration of the abdomen was done through a short transverse abdominal incision through the

* Submitted for publication November, 1949.

upper left rectus muscle and sheath. Examination disclosed a large, firm left lobe of the liver, which partially filled the left side of the upper abdomen. The lobe was 4 to 5 times normal size. Aspiration of the liver produced 30 cc. of purulent material. Cultures subsequently identified the organism to be anaerobic streptococci, susceptible to 0.2 units per cubic centimeter of penicillin. Three hundred thousand units of penicillin were instilled in the abscess, as well as 20 cc. of 30 per cent Diodrast. The area over the abscess was then packed with iodoform gauze, so that two-stage drainage of the abscess could be done. Biopsy of the liver tissue was taken and proved to be normal. After injection of radiopaque media into the cavity, roentgenograms visualized an irregular large cavity in the left lobe of the liver with finger-like projections about 7 cm. in size (see Figure 2). Immediately postoperatively he was given 100,000 units of penicillin intramuscularly every 3 hours, and subsequently 15,000 units every 3 hours for several days. The temperature did not subside but continued as high as 102.4° F. despite the sensitivity of the organism to penicillin. Four days later the packing was removed and the abscess unroofed. Packing was temporarily inserted to control considerable bleeding, and several days later this was

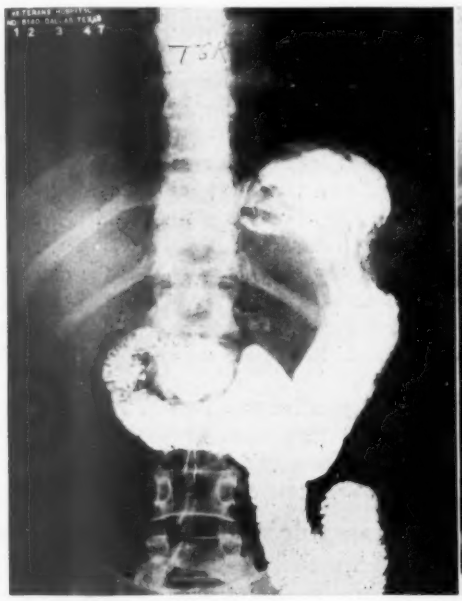

FIG. 1

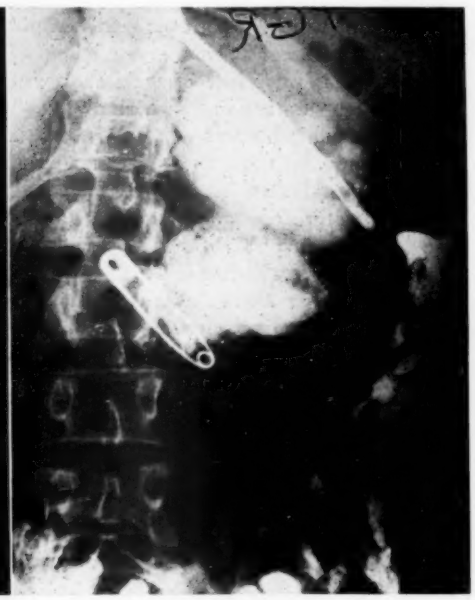

FIG. 2

FIG. 1.—(Case 1) Pressure Deformity on the barium-filled stomach (miles), with crescent-shaped lesser curve and displacement of the cardia and duodenal cap.
FIG. 2.—(Case 1) Visualization of the abscess of the left lobe of the liver with radiopaque media. The iodoform gauze packing is evident.

entirely removed and a soft rubber tube inserted. His fever rapidly subsided and the leukocytosis disappeared. The wound healed rapidly and the drainage decreased, so that complete healing had taken place in 2 months. Follow-up shows the patient to be living and without any residual difficulties.

Case 2.—*Solitary pyogenic abscess of the left lobe of the liver drained in one stage.* RBT, a white man, aged 30, was admitted to the Veterans Administration Hospital, Dallas, Texas, September 21, 1947, complaining of severe abdominal pain. Thirty hours before admission he began to experience pain in the upper portion of the left lumbar area.

The pain gradually migrated to the left upper quadrant of the abdomen, and finally, 2 hours later, localized in the epigastrium. At this point, he experienced a chilly sensation and developed a fever of 103° F. This pain was dull, worse on deep inspiration, and radiated up into the right chest, right clavicular area, and posteriorly to the right scapular area in the next 24 hours. He had anorexia but no nausea or emesis. His urine appeared darker than normal. One month before he had an episode of diarrhea associated with epigastric pain and emesis which lasted one day and subsided spontaneously. In the last 6 months or so he had had some intolerance to greasy foods.

His temperature was 99.6° F., respiration of 20, and the pulse 120 beats per minute. There was no restriction of abdominal movement on respiration. There was extreme localized tenderness in the epigastrium just inferior to the xiphoid process, and some slight tenderness on deep palpation in the right lower quadrant over McBurney's area. The pain was referred to the epigastrium upon rebound elsewhere. The liver was not palpable, but there was a sense of fullness in the upper abdomen. Roentgenograms of the chest were normal, and fluoroscopy demonstrated good movement of the diaphragm with no obliteration of the costophrenic or cardiophrenic angles. There were 16,800 white blood cells per cu. mm., of which 82 per cent were polymorphonuclear leukocytes. The erythrocyte sedimentation rate was 22 mm. in one hour. Blood and urine amylase were normal. A tentative diagnosis was made of acute cholecystitis or pyogenic liver abscess.

His temperature rose as high as 102.4° and was definitely of the septic type. He was given penicillin intramuscularly, 40,000 units every 3 hours. This was increased to 100,000 units on the fifth day after admission, when it was decided to operate upon the patient. By this time his pain had increased, the local tenderness had become extreme, and his temperature continued to have a marked swing. September 26, 1947, under general anesthesia, a small right subcostal incision was made. A perforating abscess of the left lobe of the liver was found, which was pointing anteriorly, and accounted for the extreme tenderness. The abscess was drained through a stab wound to the left of the midline. Since the abscess was leaking very little, walling off could be secured. Cultures were made of the yellow pus, but no organisms were found, despite the use of penicillinase in the culture media.

The temperature fell to normal on the first postoperative day, rose to 102.6° on the second postoperative day, but fell rapidly, so that by the fourth day he was afebrile. Three weeks later the wound had entirely healed. Subsequent investigation revealed no primary focus of this infection. Follow-up shows no residual of this abscess.

Obviously, the source of a primary pyogenic abscess of the liver can be a relatively silent lesion in the tributaries of the portal system. Indeed, it has been shown experimentally and clinically that there are two relatively unmixed columns of blood flowing side by side in the portal vein: one from the superior mesenteric vein to the right lobe and the other a mixture of blood from the splenic, gastric coronary, and inferior mesenteric veins to the left lobe.[2, 3, 4, 5] The two lobes of the liver are independent in their origin,[6] vasculature,[7, 8] and streams of portal blood.[2, 3, 5, 9] This can be demonstrated in dogs by the use of emulsified fat,[2] india ink,[3] and radioactive phosphorus.[9] Kinney and Ferrebee[4] collected 229 autopsy cases of hepatic abscess for analysis and found 136 to be bilateral, 75 right-sided, and 18 left-sided. In consideration of the points of origin of abscesses occurring in the left side of the liver only, they found direct extension of inflammation from neoplasm of the stomach, ulcer of the stomach, and abdominal inflammation in ten cases, and one each of diverticulitis of the sigmoid, ulcer of the rectum, abscess of descending colon, fistulas of ascending, transverse, and descending colon, typhoid ulcer of the ileum, neo-

plastic obliteration of the left hepatic duct, septicemia, and inflammation of the gallbladder. In general, their findings appear to substantiate the hypothesis of segregation of blood flow, except where there is an associated portal pyelophlebitis, in which case both sides may be involved. The only exception they found was a single abscess of the left lobe of the liver associated with a typhoid ulcer of the ileum. The maintenance of segregation of blood flow may be due to the shortness of the common portal trunk and the low head of pressure within it, with absence of turbulence and much mixing within the vein. In only 11 cases did the abscess appear primary in the liver, and none of these were solitary in the left lobe. Clinical experience indicates a greater number of primary abscesses of the liver than any other type. This is indicated by the series reported by Ochsner, DeBakey, and Murray,[1] in which 17 per cent of a collected series of 575 cases of pyogenic liver abscess were primary ("cryptogenic"). This variation from the autopsy incidence may be due to the impossibility of always determining the primary site in a clinical series. In general, there may be five sources of liver abscess: (1) through the portal vein and its tributaries, (2) direct extension from contiguous disease processes, (3) trauma, (4) blood borne via the hepatic arteries, and (5) through biliary channels.

In a review of the literature, with an analysis of 830 cases of pyogenic hepatic abscess and 47 personal cases, Ochsner, DeBakey, and Murray[1] found the majority of liver abscesses were amebic (74.7 per cent), occurred in males predominantly (67.4 per cent of the collected series), and the greatest age incidence was in the third to the fifth decades. Suppurative appendicitis was found to be the most frequent etiologic agent in the collected series (34.2 per cent). The organisms occurring most often were *E. Coli,* streptococci, and staphylococci, although a pure infection of anaerobic nonhemolytic streptococcus has been reported.[10, 11] The principal symptoms and signs were fever, pain and tenderness over the hepatic area, liver enlargement, chills, and jaundice. There is a leukocytosis with a proportionate increase in polymorphonuclear leukocytes. The prognosis was found to be much worse with multiple abscesses (95 per cent of their series) than with solitary abscesses (37.5 per cent). The mortality was much higher in the cases with complications (90.9 per cent) than in those without (36 per cent). These complications usually are the result of extension or rupture of the abscess into one of the adjacent viscera. The treatment consists of drainage. Where the extraserous method was possible, the mortality was found to be lower (33.3 per cent), the transpleural method higher (66.6 per cent), and the transperitoneal method the highest (72.7 per cent). It is expected that the advent of antibiotics and antibacterial drugs will materially reduce these mortality statistics. Michel and Wirth[13] report the cure of a case of multiple pyogenic abscesses of the liver due to anaerobic streptococci by the use of penicillin after surgical drainage and sulfonamides proved inadequate. It is of interest that streptomycin appears to be of little use in anaerobic nonhemolytic streptococcus infections.[12]

Although roentgenographic and fluoroscopic study are invaluable in the diagnosis of abscesses of the right lobe of the liver,[14-20] they are not nearly so characteristic or valuable in the diagnosis of abscesses of the left lobe of the liver. Case 1 (TGR) demonstrates a characteristic pressure deformity on the barium-filled stomach, described by Miles[21] (see Fig. 1). The lesser curvature assumes a crescent shape, and the cardia and duodenal cap are displaced. Beyond this, roentgenography is of little value in the diagnosis of left lobe liver abscess.

This case (TGR) admirably demonstrates the method of treating left lobe of liver abscesses, unattached to any structure, as recommended by Clairmont and Meyer.[22] This abscess was drained in two stages. The abscess was aspirated, and penicillin, as well as Diodrast, was instilled. The peritoneum was packed to produce adhesions in the first stage and to prepare for open drainage in the second stage for days later. It has been well proved that the abscess must be walled off before drainage is established in order to prevent widespread contamination and infection.[1]

These cases indicate the necessity of early and adequate drainage of these abscesses rather than continued antibiotic therapy.

SUMMARY

Two cases of solitary primary pyogenic abscess of the liver are reported.

Acknowledgment: The author wishes to express acknowledgment to Dr. John Sazama, Jr., and to Mr. Perry Boaz, clinical photographer, both of the Veterans Administration Hospital, Dallas, Texas, for the use of the roentgen ray films which were photographed for this article, and for the illustrations presented in this article.

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COMMERCIAL SOLVENTS AWARD ESTABLISHED FOR RESEARCH IN ANTIBIOTICS

The Commercial Solvents Corporation and the Society of American Bacteriologists announce the establishment of an annual award for outstanding research in the field of antibiotics.

The award, one thousand dollars and a gold medal, will be given to an individual or a group of individuals working in the Western Hemisphere who contribute to the better understanding of antibiotics. In selecting the winner of the award, particular attention will be given to the basic nature of the research upon which the award is made and its contribution to fundamental knowledge about antibiotics.

The Commercial Solvents Award will be administered by the Society of American Bacteriologists, and the recipient will be selected by a committee appointed by the President of the Society. It is expected that the first award will be presented at the annual meeting of the Society of American Bacteriologists in Chicago in May, 1951.

Editorial . . .

Changing Aspects of Surgery

As Illustrated by a Comparison of Volume 31 (1900) and Volume 131 (1950) of the Annals of Surgery

The year 1950 presents a challenge to review the progress made during the past 50 years. This has been done in the lay press and in certain fields of medicine; the present account is an attempt to assay it in surgery, as illustrated by papers in the Annals of Surgery. Volume 1 of the Annals appeared in 1885, Volume 31 in 1900, and Volume 131 in 1950. The following analysis compares the latter two volumes and the surgery of the times as reflected in them.

The general format of the Annals has changed little during these years. Volume 31 (1900) has 789 pages with a moderate number of figures, all in black and white. Volume 131 (1950) has 1002 pages with many figures, mostly in black and white, but which also include a few in color. Volume 31 includes the Proceedings of the New York Surgical Society and of the Philadelphia Academy of Surgery in abstract form in most of the issues. Volume 131 includes, aside from independently submitted papers, the full papers of the Southern Surgical Association Proceedings. At the present time, the even-numbered volumes (*e.g.*, Volume 130) contain the Proceedings of the American Surgical Association; Volume 30 does not. Volume 31 has a combined subject and author index, book reviews, editorials, and abstracts of the literature. Volume 131 has added separate subject and author indexes and has dropped the abstracts of the literature. There are 47 original articles in Volume 31 (not counting abstracts and proceedings in abstract form) while Volume 131 has 97 original articles including the papers of the Southern Surgical Association. Judging by modern standards, the general format, printing, and quality of the illustrations have improved markedly.

Volume 31 began with an article by Harvey Cushing, then 30 years of age, entitled: "The Employment of Local Anesthesia in the Radical Cure of Certain Cases of Hernia, with a Note upon the Nervous Anatomy of the Inguinal Region." This paper is more concerned with the second aspect of the title, the nervous system, than with the first, the technic of herniorrhaphy. It is of interest that Cushing's article is the only experimental article in Volume 31 and that it presages the future by pertaining to Cushing's later special field of work. Fifty years later, Volume 131 begins with another clinical experimental study by J. Garrott Allen, 37 years of age, and his associates, but this time it is not the only one in the volume.

The fields of surgery covered by the two volumes make an interesting study which is summarized in Table I. Any classification of this type is

difficult, but an attempt has been made to use the same standards for analysis of each of the two volumes. The recent influence of special journals affects an accurate comparison; the decrease in the number of articles on urology from three to one, as seen in Table I, may merely reflect the influence of the *Journal of Urology,* first published in 1917, which contains some 120 articles on the subject per volume. On the other hand, certain fields have shown an increased representation in the Annals, as, for example, articles on thoracic surgery, which have increased despite the existence of a quarterly specialty

TABLE I.—*Shifting Emphasis Concerning Phases and Fields of Surgery and Development of New Interests in Surgery (As Illustrated by Articles in Annals of Surgery, 1900-1950).*

Field of Surgery	Number of Articles	
	Volume 31 1900	Volume 131 1950
Principles and General Problems of Surgery	12	21
Anesthesia	0	1
Surgical Diagnosis	1	0
Surgical Anatomy	0	2
Preoperative and Postoperative Care	1	6
Technic	1	1
Wound Healing	1	1
Surgical Infections	5	2
Tumor Surgery	1	5
Trauma (excluding fractures)	2	2
Surgical Training	0	1
Regional Surgery	35	76
General Surgery: Abdomen	9	35
Hernia	4	1
Thyroid and parathyroid	0	2
Breast	0	3
Peripheral vascular	1	6
Miscellaneous	2	3
Neurosurgery	2	6
Thoracic Surgery	0	12
Plastic Surgery	0	2
Urology	3	1
Gynecology	2	2
Orthopedics: Fractures	4	1
Amputations	3	0
Other	5	2
Totals	47	97

journal. Such differences may depend on the changing provinces of general surgery (vid. Churchill, Editorial: "General Surgery," Annals of Surgery 131: 127, 1950).

Differences in trend that are of interest include an increase in the number of papers on preoperative and postoperative care, tumor surgery, abdominal surgery, breast surgey, surgery of peripheral vascular disease, neurosurgery (despite a specialty journal) and thoracic surgery (including the heart). Important subjects or organs which received no attention in the 1900 volume are represented by several articles in 1950 (*e.g.,* heart, pancreas, thyroid, para-

thyroid, and breast) and the subject of the training of surgeons is the topic of one paper in 1950 and of none in 1900. It must, in all fairness, be mentioned that certain of these phases are cited in the proceedings or literature abstracts, even though they are not covered in the original articles in the 1900 volume. The abstracts are omitted from this study so that the comparative analysis of the two volumes might not be carried to too fine a point. Of further interest, the 1900 volume contains two articles representing arm-chair philosophy, but there are none in the 1950 issue. The balance between emphasis on general problems and on regional surgery has remained about the same (25 and 22 per cent, respectively, of the total being general problems). On the other hand, many of the clinical articles include more laboratory studies than those in 1900, as would be expected.

TABLE II.—*Increased Emphasis on Experimental Approach in Surgery (As Illustrated by Articles in Annals of Surgery, 1900-1950).*

	Volume 31 1900		Volume 131 1950	
Type of Article	No.	%	No.	%
Clinical observations, case reports, and literature reviews	46	98	83	86
Animal experimental research	0	0	5	5
Clinical experimental research	1	2	9	9
Totals	47	100	97	100

The second feature of the comparative study lies in the increased interest in animal and clinical experimental research that has occurred over the period of 50 years as indicated in Table II. Again, comparison is difficult because the borderlines between the types of articles are subject to variation of interpretation. In every way possible, the same criteria are used in classifying the papers in each volume. The increase in research journals (*Proceedings of the Society for Experimental Biology and Medicine, Journal of Clinical Investigation*, etc.) has undoubtedly siphoned off many experimental papers so that the relative increase in the Annals may represent only part of the trend. Also, a purely surgical journal will probably always be of necessity weighted clinically.

Qualitatively, the research papers in 1950, either animal or clinical, show an increase in utilization of the methods of chemistry and physics. Many of the articles show the trend towards group or co-operative research, and the apparent expenditures of money involved are to some extent a product of industrialization of the age more than of an independent advance in science. The small unit, individual type of research project has not entirely been abandoned, fortunately, and there are several excellent papers representing this type of work in the 1950 volume.

The added perspective afforded by the passing of 50 years gives increased interest to many of the papers. Such is true of the article by John Bruce

Harvie of Troy, New York, entitled "Case Report of Recovery after Gastrectomy for Carcinoma." The same applies to the paper by Bullitt of Louisville, "Report of a Case of Actinomycosis Hominis of the Lungs," without operation, but with an autopsy by "Professor Flexner." It is a fascinating pastime to peruse the volume of 50 years ago and to speculate about the men who wrote its papers. The editors of Volume 31 deserve credit for selecting Dr. Cushing's paper, for its author is better known today than he was in 1900. The same applies to other authors in the former volume whose names are well recognized today. This group includes, aside from Cushing (then of Baltimore), such men as Berg (New York), Erdmann (New York), Fowler (New York), J. Shelton Horsley (then of El Paso), Freeman (Denver), Pilcher (New York), Richardson (Boston), Warbasse (New York) and Royal Whitman (New York). The 1950 volume also includes the names of well-known surgeons. Both volumes serve as a record of outstanding surgical advancements of their times. Real discoveries are lasting. Those who make the discoveries will have a permanent place in surgical history even though methods or technics are altered or improved. We can be confident that just as the outstanding contributors to Surgery of 1900 are still known today, so the original workers of 1950 will be similarly recognized in the year 2000.

HENRY N. HARKINS.

BOOKS RECEIVED FOR REVIEW

LOUIS J. GARIEPY	*Saw-ge-Mah* (*Medicine Man*). Northland Press, St. Paul, Minnesota, 1950. (326 pages, 3.00)
JAMES MORONEY	*Surgery for Nurses.* The Williams and Wilkins Company, Baltimore, Maryland, 1950. (619 pages, 6.00)
WALTER BUCHLER	*Parkinson's Disease.* (Advice and Aid for Sufferers from Parkinson's Disease and Other Physical Disabilities.) Walter Buchler, London, England, 1950. (75 pages, 1.00 paper cover, 2.00 cloth binding)
KENNETH W. STARR	*The Causation and Treatment of Delayed Union in Fractures of the Long Bone.* Butterworth & Co., Ltd., London, England. (The C. V. Mosby Company, St. Louis, Mo.) 1947. (201 pages, 9.00)

BOOK REVIEWS

THE SURGICAL TREATMENT OF FACIAL INJURIES. By V. H. Kazanjian and J. M. Converse. The Williams and Wilkins Co., Baltimore, 1949, $10.00.

This book is based on the experience of the authors in two world wars, and in practice. It presents their conception of, and procedure in, the treatment of facial injuries. Following chapters on the anatomy of the face, and general principles, the various problems presented by the many injuries that can affect the face are described. Since these are admittedly from personal experience, and no man can know everything, the text is not comprehensive. No mention, for instance, is made, critically or otherwise, of the use of sand papering in the treatment of powder marks, nor of Kirschner wire transfixion in the treatment of fractures of the mandible. Thus the book cannot be used as a final reference. It does thoroughly and lucidly present the management of facial injuries as done by them. There is, therefore, a great deal of valuable information to aid both the general and the plastic surgeon. This volume will be of help to the general surgeon called upon from time to time to treat a facial injury, and will prove a veritable mine of information for the plastic surgeon. It is a must for the plastic surgeon's library.

STUART D. GORDON, M.D.

SURGICAL AND MAXILLOFACIAL PROTHESIS. O. E. Beder. King's Crown Press Columbia University, New York, 51 pages, $3.00.

This is a concise, 51-page treatise on the preparation of prosthetic appliances for use about the face and head. Its title is as misleading as the definition of "surgical and maxillofacial prosthesis" is incorrect. Surely surgical prostheses are used in many parts of the body and the art of making them not entirely a branch of dentistry!

This manual will be of value to a dentist, and his technician, attached to a plastic surgical unit. Technical steps are detailed and definite. A chapter on shields in radiation therapy is included.

STUART D. GORDON, M.D.

PRINCIPLES AND PRACTICE OF PLASTIC SURGERY. A. J. Barsky, M.D. The Williams & Wilkins Co., Baltimore, 1950.

This compact volume purports to be a practical guide to the practice of plastic surgery. Fundamental principles are discussed in the first six chapters, which include an excellent and succinct presentation of the psychologic aspects of the specialty. The remaining portion of the book is devoted to a practical discussion of most of those conditions treated by a plastic surgeon.

These conditions are thoroughly described and definite treatment, based on the author's experience, recommended. Such recommendations are, on the whole, excellent. The reviewer, for example, could not be in more complete agreement with the consideration of external fixation in the treatment of mandibular fractures. An adequate, though not exhaustive, bibliography follows each chapter.

In the opinion of the reviewer the author has admirably attained his stated objective. This volume will prove invaluable to the trainee in plastic surgery, and be extremely useful to the plastic surgeon as a reference, and as a starting point for more detailed study. This is a text which one can recommend without reservation.

STUART D. GORDON, M.D.

INDEX TO VOLUME 132

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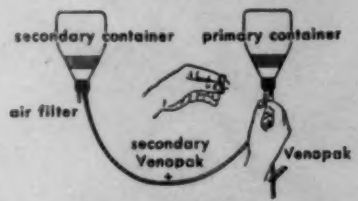

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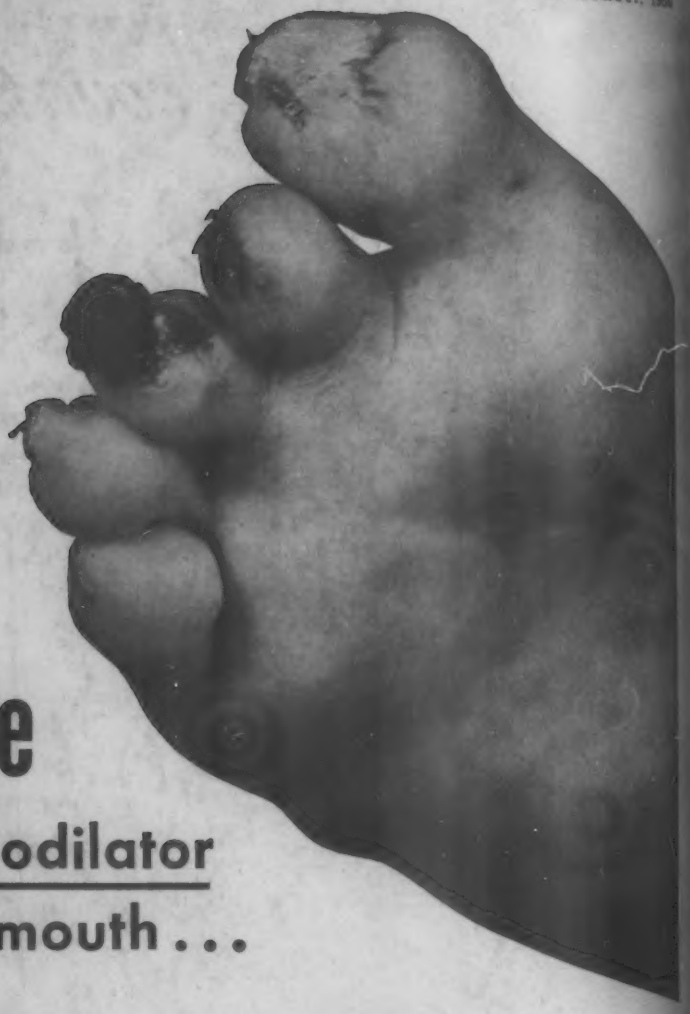

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Comprehensive literature on request.

1. Rogers, Max P.: J.A.M.A., May 21, 1949
2. Wyatt, Bernard L.: Ann. West. Med. & Surg., Aug. 1949
3. Grimson, Marsoni, Reardon & Hendrix: Ann. Surg., 127:5, May 1948

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